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THE
LARYNGOSCOPE.

VOL. LXIII SEPTEMBER, 1953 No. 9

PARALYTIC STENOSIS OF THE LARYNX.

A Modification of the Intralaryngeal Approach to Arytenoidectomy.*

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PURPOSE.

It is the purpose of this paper: *1.* to discuss the effect of bilateral recurrent laryngeal nerve paralysis on the musculature of the larynx when the nerves are inadvertently injured during a thyroidectomy; *2.* to review the various methods which have been utilized to combat the resulting paralytic stenosis, and *3.* to present a modification of the intralaryngeal arytenoidectomy for the relief of bilateral abductor paralysis of the vocal cords.

EFFECT OF BILATERAL RECURRENT LARYNGEAL NERVE PARALYSIS ON THE LARYNGEAL MUSCULATURE.

According to Cunningham's Textbook of Anatomy,[20] the nerve supply of the larynx is derived from the superior laryngeal and recurrent branches of the vagus nerve. The superior laryngeal nerve is the sensory or receptor nerve from the mucous membrane of the larynx, and also gives off a branch which furnishes motor or effector fibres to the cricothyroid

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muscle. All of the other muscles of the larynx receive their motor or effector nerve fibres from the recurrent laryngeal nerve.

All of the intrinsic muscles of the larynx are composed of striated muscular fibres and, with the exception of the cricothyroid muscle, all lie under cover of the thyroid cartilage. The abductor muscles of the larynx consist of only one pair, the posterior cricoarytenoid muscles, which, during respiration, abduct the vocal cords and are sometimes called the openers of the glottis. The adductor muscles, or closers of the glottis, include *1.* the interarytenoid (transverse and oblique); *2.* the thyroarytenoid, and *3.* the lateral cricoarytenoid. The tensors of the vocal cords are the cricothyroid muscles.

Since both adductor and abductor muscles are innervated by the recurrent laryngeal nerve, severance or serious injury to the nerve causes a lower motor neuron or flaccid type of paralysis of both groups of muscles. The recurrent nerve is different from other nerves in that under normal conditions it conveys antagonistic impulses; that is, impulses which abduct the vocal cords during respiration and adduct them on phonation. The mechanism responsible for the positioning of the vocal cords, after both recurrent nerves have been injured, has been a matter of considerable interest and speculation.

In 1881, Sir Felix Semon,[21] of London, published his classical work on paralysis of the larynx, in which he stated: "There is a distinct proclivity of the abductor fibres of the recurrent laryngeal nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of central or peripheral injury or disease of the recurrent nerve." In order to prove this statement, Semon presented 22 case reports, including clinical observations and postmortem examinations, to show that isolated paralysis of the posterior cricoarytenoid muscles was earlier than that of their antagonists. Semon concluded that the abductor nerve fibres are more susceptible to disease or injury than are the adductor nerve fibres in the recurrent laryngeal nerve, and that when an organic lesion or injury affects the nerve, the abductor muscles are first to

be affected, causing the vocal cords to remain in a state of adduction; however, as the disease or injury progresses, the adductor fibres become more involved and the final picture is that of the cadaveric position.

New and Childrey[18] do not feel that Semon's law can be substantiated. They observed that following injury to the recurrent laryngeal nerve a large percentage of vocal cords are in the median line position and that a small percentage are in the cadaveric position. Follow-up data revealed that either the vocal cord returns to its normal function, or it pulls to the median line within a few months and remains there. No instance in which the vocal cord changed from the median line to the cadaveric position was observed.

A study of 217 cases of paralysis of the vocal cord was made at the Mayo Clinic. According to Dilworth,[6] the arytenoid muscles receive a few fibres from the superior laryngeal nerve as well as their supply from the recurrent nerve. He reported that if the recurrent nerve becomes paralyzed the vocal cord assumes the median line position in 90 per cent of cases; but, in a small percentage of cases, the vocal cord is in the cadaveric position. In the latter group, the affected cord that does not return to function, pulls to the median line and is tensed by the action of the arytenoid and the cricoarytenoid muscles which receive innervation from the superior laryngeal nerve. In 26 cases, patients were examined after intervals varying from a few weeks to a number of years. The paralysis in these patients was due to various causes. It was concluded from this study of 26 cases that, in general, a partially paralyzed vocal cord (so-called abductor type) may completely recover function; a vocal cord fixed in the median line may completely recover, but usually remains in this position; and a vocal cord fixed in the cadaveric position may complely recover, but usually swings to the median line within a few months and remains there.

Lederer[13] points out that the subject of motor dysfunction of the larynx is still a controversial one. He states that the literature on neuropathology of the larynx reveals two oppos-

ing schools. One accepts Semon's "law" (that the abductor muscles, posterior cricoarytenoid, of the larynx show a greater tendency to motor impairment than the adductors, thyroarytenoids, transverse and oblique) as satisfactorily explaining all of the neuropathologic motor reactions of the larynx. The other group insists that no such law has been established. According to Lederer, "There is unquestionable evidence incompatible with Semon's law." Many laryngologists believe there are other mechanisms involved, undoubtedly other nerves or muscles, that keep the vocal cord or cords adducted despite the destruction of the major or recurrent laryngeal nerve supply.

Numerous theories have been advanced to explain Semon's law. Semon[21] felt that there is a physiologic weakness of the muscles, nerves and center subserving the function of abduction. Others hold that *1.* the adductor nerve fibres are situated in the periphery of the recurrent laryngeal nerve where they are affected by any noxious process; *2.* pressure on the recurrent nerve first stimulates the abductor fibres, and then wears them out, leaving the adductor fibres intact; *3.* the abductor muscles are like other extensor muscles in the rest of the body, and are more susceptible to trauma and disease than flexor or adductor muscles; *4.* the abductor muscles are of later phylogenic development than the adductor muscles and biologically weaker or more susceptible to injury. According to Jackson and Jackson,[10] spasticity explains the sequence of the midline position in cases of bilateral paramesial position and in at least some of the cases of bilateral incomplete paralysis.

According to Blalock and Crowe,[3] histologic studies of recurrent nerves removed at necropsy from patients who had clinical evidence of impaired motility of a vocal cord as a result of local pressure or stretching of the nerve failed to show any definite zone in which the degenerated fibres lie, and the experimental studies on regeneration showed that the new fibres appeared at random throughout the nerve.

In 1931, Hoover[8] stated that, at the Lahey Clinic, the most common cause for bilateral abductor paralysis was direct

trauma or operative injury by surgical procedures in the region through which these nerves pass, or direct pressure by, or involvement in a pathological condition in these regions. In 17 of the 18 patients seen by Hoover, the cause of bilateral paralysis was related to thyroid conditions. Fourteen instances of bilateral abductor paralysis followed thyroid operations; one followed a malignant condition of the thyroid; one, syphilis of the thyroid; one, a thyroid condition and a tumor of the neck; and one followed a fracture of the larynx, high tracheotomy and perichondritis. Mention was also made that laryngeal paralysis due to benign enlargement of the thyroid was very rare. Hoover states that in bilateral abductor paralysis, the cords may assume the cadaveric position, but no cases were seen at the Lahey Clinc. Contrary to the impression most often received from textbooks and teachers of laryngology, the paralyzed cord more frequently lies in the median position than in the cadaveric position in instances of unilateral paralysis.

METHODS UTILIZED TO COMBAT PARALYTIC LARYNGEAL STENOSIS.

It was natural for the early clinicians, confronted by double abductor paralysis, to investigate the possibility of reuniting the severed inferior laryngeal nerves or re-establishing innervation by some type of anastomosis. In 1924, Frazier[7] reported his observations on the effect of suturing the descendens hypoglossi to the recurrent laryngeal nerve. In seven instances where a satisfactory suture was effected, function apparently returned in three patients. The others were operated upon too recently to warrant a report. In this study, the suitability of a given case for neuroplasty of recurrent nerve was carefully considered as to *1.* the duration of the paralysis, *2.* the motility of the cricoarytenoid joint, and *3.* the existence of an undamaged segment of nerve.

Sir Charles Ballance,[2] of London, carried out experiments on goats and monkeys in an effort to determine the value of the anastomosis of the divided recurrent laryngeal with another nerve. Examination of the larynx of animals in which

the recurrent nerve was severed, at varying periods up to 200 days after the operation, revealed no respiratory movement of the vocal cord. At first, a typical cadaveric position was said to occur and the inner border of the vocal cord was concave; but after from six to eight weeks from the time of operation, the inner border of the immobile vocal cord became straight and tense. There was also a tonus about the paralyzed cord which had not been present before. The change of tone occurred whether recurrent-laryngeal-vagus or recurrent-laryngeal-descendens-noni had been done. Stimulation proximal to and at the site of anastomosis caused abduction of the vocal cord, but the excursions of the cord were not so wide as on the opposite or healthy side. After stimulation was carried out for some time, strong adduction occurred. It was concluded that adduction was the result of fatigue of the abductor muscle. Ballance also observed that the associated emotional movements of the face required a long period of re-education by voluntary effort before they were perfect. Since the movements of the vocal cords are subconscious acts, a similar period of time might be required for the recovery of the associated movements of the vocal cords. It was thought that the difference might lie in the possibility of treatment. In the face muscles, re-education of the associated movements of the two sides of the face is practicable by voluntary effort; but any such re-education is impossible in the muscles controlling the movements of the vocal cords; hence, it was deduced that if the patient held his breath over a sufficient period of time, anoxia would be created and the problem of re-education might be overcome.

Blalock and Crowe[3] studied recurrent nerve regeneration in dogs and presented both physiological and histological evidence of regeneration following an anastomosis of the recurrent laryngeal to the following nerves: *1.* the upper root of the phrenic (end to end); *2.* the phrenic (end to side); *3.* the vagus (end to side); and *4.* the descending hypoglossal (end to end). When the recurrent nerve was anastomosed to the descending hypoglossal, movement of the cord usually returned earlier than it did in the other experiments, and the excur-

sion was thought to be greater. Rapid regeneration occurred not only when the ends of the two nerves were connected loosely with a silk thread, but also when the distal end of the recurrent was placed into a slit in an adjacent sound nerve. Return of movement of the paralyzed cord was noticed slightly earlier when the descending hypoglossal nerve was used. If it were not for the small size and position of the upper root of the phrenic nerve, it would seem the most ideal for anastomosis because the functions of the larynx and the diaphragm seem more closely related than those of the larynx and the neck muscles that are supplied by the descending hypoglossal. Colledge,[5] working with Ballance,[2] reports a case in which movement returned after an anastomosis of the recurrent laryngeal nerve into a slit in the phrenic nerve. According to Kelly,[11] nerve anastomoses were unsuccessful because it was found that the impulses arriving as a result of suture or anastomosis went not only to the abductor but also the adductor muscles, producing spasmodic movements of the vocal cords. Clerf[4] stated that he had an opportunity to observe a number of patients who had an anastomosis of the recurrent nerve, but never saw a single satisfactory result.

When it became apparent that anastomosis of the injured recurrent nerve with other nerves could be accomplished but failed to produce the desired result of abducting the vocal cord, attention was directed toward operations on the vocal cord which would alleviate the dyspnea and permit decannulation. One of the earliest recorded operations of this type was reported by Baker[1] in 1916. In this instance of bilateral abductor paralysis, a successful airway was obtained by the removal of one vocal cord and arytenoid by a laryngofissure.

In 1922, Jackson[9] reported what he termed a new operation for the cure of goitrous paralytic laryngeal stenosis. The procedure was termed ventriculocordectomy, and the technique was as follows: The anesthetized larynx was exposed with a laryngoscope and the ventricular band was elevated and forceps applied so that the floor of the ventricle and part of the mucosa of its outer wall could be removed by one clip. Dr. Jackson added that although excision of the cord and

ventricular floor could be executed by laryngofissure, this seemed a more formidable operation to the patient. In 11 of the 18 cases of ventriculocordectomy reported, the operation failed because it was discovered that the paralytic stenosis was complicated by cicatricial stenosis due to high tracheotomy, faulty postoperative care, improperly shaped or fenestrated cannulas. It was concluded that no patient should have a ventriculocordectomy unless a 7 mm. bronchoscope would readily pass through the larynx. Of the 18 patients having ventriculocordectomy, the seven that were uncomplicated by cicatricial stenosis were afforded satisfactory relief of dyspnea by this procedure alone.

When it became apparent that cicatricial tissue obviated the desired result in ventriculocordectomy, Hoover,[8] in 1932, suggested that by submucous resection of the vocal cord, one would eliminate the exuberant granulation and could expect an adequate airway to be maintained.

About the same time, Loré[14] developed a similar operation. Loré exposed the larynx by means of a laryngofissure and detached the vocal cord, except for its attachment, anteriorly. The arytenoid cartilage was dissected free and removed. The vocal cord was pulled forward and the subjacent tissues removed down to the perichondrium, then returned to its original position, except posteriorly, where the free end was more external because of the absence of the arytenoid. The vocal cord was then sutured in this position.

In 1939, King[12] presented a new operation for the relief of bilateral paralysis of the vocal cords. All previous procedures have proved unsatisfactory, but the King operation has stood the test of time. King originally hoped actually to restore function of one of the vocal cords by an orthopedic operation on the cricoarytenoid joint. The basic principle of the original operation consisted of disarticulating the cricoarytenoid joint and attaching the cut end of the omohyoid muscle to the arytenoid. Since it had been shown that the omohyoid contracts on inspiration, it was hoped that there would be sufficient pull exerted on the arytenoid cartilage by the contrac-

tion of the omohyoid to abduct the vocal cord and allow normal respiration. It was later determined that the success of the operation was not due to the substitution of a viable accessory muscle of respiration for the inactive posterior cricothyroid muscle. As pointed out by Morrison,[15] under conditions of forced respiration, the action of the muscle could not contribute more than 1 or, at the most, 2 mm. of additional airway. During normal respiration its action is negligible.

In 1941, Kelly[11] published a report on two cases of bilateral abductor paralysis in which the arytenoid cartilage was removed by an extralaryngeal approach through a small window created in the wing of the thyroid cartilage. Kelly reasoned that if he could perform an arytenoidectomy through the wing of the thyroid cartilage he might accomplish three things: *1.* Destroy the tension of the cord on the side on which the operation is performed, which is caused by the action of the cricothyroid muscle, and allow the cord to fall away from the midline of the larynx. *2.* The space created by the loss of the arytenoid cartilage would be obliterated by nature and thereby cause an atrophy of the muscles on that side which would further increase the area between the cords posteriorly. *3.* The position of the anterior third of the cord would be slightly altered in relation to the side which was not operated upon, and in that way would preserve, to a reasonable extent, a proximity of the vocal cords which would give a fairly serviceable voice.

In 1946, DeGraaf Woodman[23] reported a case of bilateral abductor paralysis of the larynx in which the arytenoid cartilage was removed by an approach posterior to the thyroid cartilage. The posterior approach to external arytenoidectomy eliminates the necessity of making a window through the thyroid cartilage or removing half of the cartilage as recommended by Orton.[19] The vocal cord is drawn laterally and sutured to the inferior cornu of the thyroid cartilage.

Since the preliminary report, Woodman[24] has reported the results of 23 cases where the "open approach" technique was used. Only two patients, or about 9 per cent, were wearing

tubes, and this was said to be no failure of the surgical procedure but was the result of the mental condition of both patients. Some of the 23 cases were from the services of other laryngeal surgeons at the Columbia Presbyterian Medical Center and Dr. Fletcher Woodward, of Virginia, and Dr. Frances LeJeune, of New Orleans.

INTRALARYNGEAL APPROACH FOR ARYTENOIDECTOMY.

In 1951, I was asked to see a patient who had marked laryngeal obstruction. An examination revealed both vocal cords approximated at the midline with an extremely small glottic chink, posteriorly. A tracheotomy was performed, and it was apparent that eventually one of the operative procedures for the relief of bilateral abductor paralysis would be required if the patient was to be decannulated. When one reviews the literature on this subject, it becomes evident that many operative procedures have been devised, but relatively few have stood the test of time.

In a personal communication, Dr. V. E. Negus,[17] of London, stated that he was in favor of permanent tracheostomy, using a specially designed tracheostomy tube. He stated further that "patients with a permanent tube suffer practically no disability provided they do not go swimming. Their voice is clear and their respiration free."

In 1948, Thornell[22] recommended the removal of the arytenoid cartilage by means of the intralaryngeal approach, carried out under intravenous pentothal sodium anesthesia, through the Lynch laryngeal suspension apparatus. An incision of 1 cm. is made over the superior surface of the arytenoid cartilage and extended anterolaterally into the aryepiglottic fold. Submucous dissection and removal of the arytenoid cartilage is then carried out. Three cases were presented in the original report, and good results were obtained by all three patients. In discussing Dr. Thornell's operation, Dr. Lewis F. Morrison[16] stated that, "if the results are equal to those obtained by the extralaryngeal approach, that a procedure has been advanced that is less difficult and less time

consuming than any previous method. In addition, it should be less of a physical and financial burden on the patient." In the same discussion, Morrison stated that he had performed the King operation on 46 patients and that all had an adequate airway and only three had poor voices. Kelly,[11] Woodman[24] and Thornell[22] have established the fact that an arytenoidectomy would relieve dyspnea and preserve the voice in a large majority of patients with bilateral abductor paralysis; yet, all three offer a different approach for removal of the arytenoid cartilage.

After dissecting several arytenoid cartilages from cadavers, it was found that disarticulation and complete removal of the cartilage without fracture was, at times, somewhat difficult. Having utilized the Lynch suspension laryngoscope only for biopsies and realizing the technical difficulties of the Kelly operation, necessitating dissection through a window (three-eighths-inch) in the thyroid lamina, I concluded that the most easily accessible approach for arytenoidectomy would be by the midline thyrotomy incision.

Noting the rapid healing and epithelialization following laryngofissure for the removal of cordal cancer when the antibiotics are used, and encouraged by the fact that Thornell[22] had successfully employed the intralaryngeal technique, I felt that an arytenoidectomy could be safely and more easily performed through the midline thyrotomy incision. It also seemed that an intralaryngeal arytenoidectomy, performed through the laryngofissure incision, would offer other advantages. By retracting the thyroid alae, excellent exposure of the arytenoid area would be afforded, dissection and disarticulation could be easily accomplished, hemostasis readily controlled, and, finally, the mucosal incision could be carefully approximated and sutured.

Outline of Technique for Intralaryngeal Arytenoidectomy:

1. A midline incision is made over the thyroid cartilage. the overlying fascia and muscles are separated by sharp and blunt dissection.

2. The thyroid cartilage is divided in the midline.

3. The thyroid alae are retracted, and 0.5 cc. of 1 per cent novocaine with adrenalin is injected into the mucosa over the arytenoid.

4. An incision, 0.5 cm. in length, is made in the mucosa, directly over the arytenoid cartilage.

5. The arytenoid cartilage is dissected.

6. The cricoarytenoid joint is disarticulated and the arytenoid cartilage is removed.

7. Hemostasis is accomplished with pressure, electrocoagulation, or ligation.

8. The mucosa is closed with a 0000 absorbable surgical suture. The thyroid alae are approximated, and the overlying muscles and skin are sutured.

CASE REPORTS.

Case 1: On Aug. 11, 1950, the patient, a white woman, age 51, was seen as an emergency. She had severe dyspnea and laryngeal stridor. Her husband stated that the patient had a thyroidectomy elsewhere in 1938, and was quite hoarse for three months following surgery. Since that time she had dyspnea on exertion, and for two months the dyspnea had been severe.

On examination, paradoxical respiration was noted. Roentgenograms of the chest revealed a fracture of the third, fourth, fifth, sixth, seventh and eighth ribs in the posterior axillary line. Both vocal cords were fixed in the midline and approximated so as to seriously obstruct the air passage. The cause of the fractured ribs was presumably due to the forced respiratory efforts of the patient as there was no history of trauma.

An immediate tracheotomy was performed. When the tracheotomy tube was inserted, breathing ceased. A bronchoscope was inserted, oxygen therapy and artificial respiration were instituted and after two minutes respiration recurred. The patient reacted satisfactorily and had an uneventful convalescence. The ribs healed satisfactorily in four weeks. On Sept. 25, an arytenoidectomy was performed on the right side, using the midline thyrotomy technique. On Oct. 24, the tracheotomy tube was removed and the tracheotomy stoma was closed. Following the arytenoidectomy, the patient gained 27 pounds (12.2 kgm.), has had no dyspnea, even on exertion, and the voice is excellent.

On Sept. 3, 1952, the patient was contacted by telephone. She stated that she was able to carry on her household duties satisfactorily, and had experienced no dyspnea since the arytenoidectomy. Her voice was still excellent.

Case 2: The patient, a 58-year-old white woman, was seen in my office on May 25, 1950. She complained of dyspnea on exertion following subtotal thyroidectomy four months previously. An examination of the larynx revealed both vocal cords near the midline with a very narrow glottic chink, and the cords failed to abduct on respiration. The voice was coarse and husky. Recommendations at that time were, that if dyspnea became worse, the patient would require a tracheotomy.

Because the dyspnea grew progressively worse, on Aug. 30, 1950, a tracheotomy was performed. On Sept. 5, 1950, a right arytenoidectomy was performed, using the laryngofissure technique. After about 10 days, the postoperative edema subsided, and the patient appeared to have an adequate airway; however, she had a chronic, productive cough which had been present prior to surgery, and during paroxysms of coughing, experienced some difficulty in breathing when the tracheotomy tube was occluded. On Dec. 15, 1950, it was decided that the tracheotomy tube might be aggravating the cough. Decannulation was carried out, and the cough improved. There was no respiratory difficulty resulting from decannulation. On April 16, 1951, examination revealed an adequate glottic chink of 3.5 mm., posteriorly; and the patient was able to carry on her work without respiratory distress. The quality of the voice was not so good as it had been prior to surgery but was plainly audible and serviceable.

On Sept. 3, 1952, the patient returned for observation. She stated that she had no trouble breathing—except when she mowed the lawn. She has been doing her housework, and stated that she felt well. Her voice was a little coarse. There appeared to be a glottic space of approximately 3 mm. at the posterior extremity of the larynx.

Case 3: I examined the patient, a white woman, age 37, on Nov. 9, 1951. She stated that 18 months previously, a thyroidectomy had been performed elsewhere. For three months following the operation, she had been quite hoarse. Her voice gradually improved, but respiration had become more difficult; and during an upper respiratory infection, an emergency tracheotomy was performed. After the laryngeal infection subsided, the tracheotomy tube was removed; however, at the time of examination, the patient was unable to perform her duties as a housewife because of dyspnea upon exertion. The patient had been told that she made a rather loud respiratory noise during sleep.

On Nov. 14, 1951, using local anesthesia, a tracheotomy was performed, and a midline incision was made over the thyroid cartilage. The overlying ribbon muscles were retracted, the thyroid cartilage was incised, and the thyroid alae were retracted. Novocaine (1 cc. of 1 per cent) with alidase and 1:10,000 adrenalin was injected into the right aryepiglottic fold over the arytenoid cartilage. The cartilage was then dissected free and removed. The mucosal incision was sutured with interrupted 00000 absorbable suture. Laryngeal edema and operative reaction subsided satisfactorily and the patient was allowed to return home on the sixth postoperative day. A letter from an otolaryngologist, dated Dec.

31, 1951, stated: "The right vocal cord is now nicely abducted and the airway is quite adequate. Decannulation has been carried out. This patient's result appears to be excellent, and we shall continue to follow her from time to time."

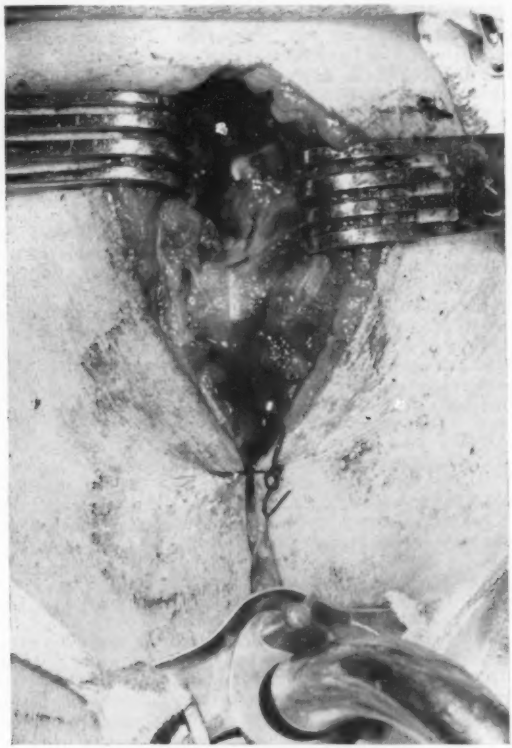

Fig. 1. Exposure of left arytenoid cartilage through laryngofissure incision (Case 4).

On Aug. 13, 1952, the patient returned for observation. She stated that the "crowing" respiration at night had ceased, and that she was able to do routine housework; although if she exerts herself too much, such as climbing stairs, she has some dyspnea. She has had an attack of laryngitis but recovered nicely and had no severe respiratory distress. The quality of the voice was fairly good. Examination revealed a space of 4 mm. in the arytenoid area.

Case 4: On May 29, 1952, I was asked to see this patient, a 38-year-old white woman, whose chief complaints were dyspnea on the slightest exertion and the persistence of a crowing type respiratory noise during sleep. She had lost 15 pounds during the past six months. The patient stated that she had a thyroidectomy seven years previously. Examination

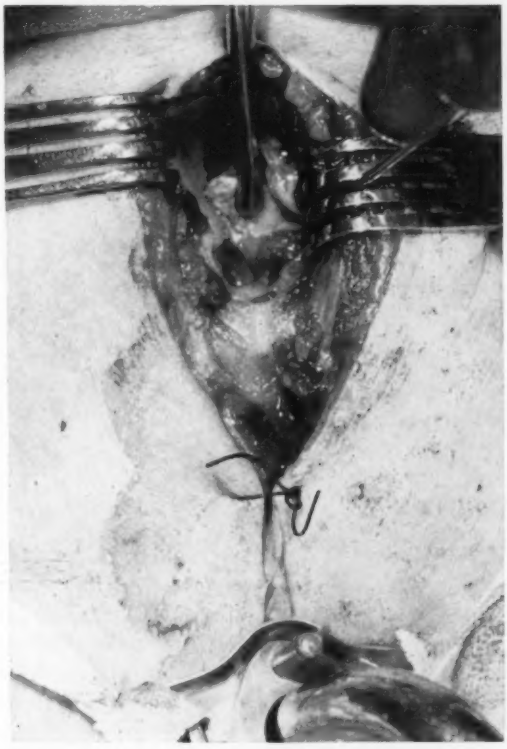

Fig. 2. Dissection of the left arytenoid cartilage (Case 4).

revealed the right vocal cord in a paramedian position near the midline and the left vocal cord in the midline position. Neither cord moved during respiration.

On June 2, 1952, a tracheotomy was performed, using local anesthesia. The patient was then given intravenous pentothal sodium anesthesia, and a left arytenoidectomy was performed by the laryngofissure tech-

nique. One week later, the patient was permitted to return home to be under the care of her local ear, nose and throat physician. He decannulated the patient on June 24, 1952, and reported that the laryngeal aperture appeared adequate and the voice was satisfactory.

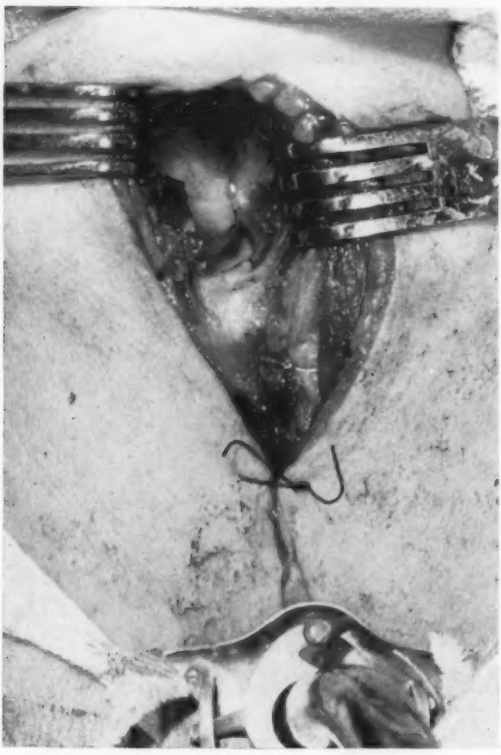

Fig. 3. The left arytenoid cartilage has been removed and the mucosa has been sutured (Case 4).

On Aug. 18, 1952, the patient returned and the following note was recorded: "Patient returns for postoperative observation of an arytenoidectomy. She states that she no longer has the crowing respiration at night, has adequate breathing space, does her housework satisfactorily, and her voice is very good, there being just a mildly coarse quality present. She is well satisfied with the results of the arytenoidectomy."

Fortunately, bilateral abductor paralysis is not a common complication of thyroidectomy. Refinements in surgical technique, the meticulous dissection and identification of the recurrent and inferior laryngeal nerves, have decreased the incidence of nerve injury. All patients undergoing thyroid surgery should have pre- and postoperative laryngeal examinations. Patients who have a bilateral abductor paralysis of the larynx resulting from thyroidectomy should be advised as to the nature and potential seriousness of the condition, and should be under the care of an otolaryngologist.

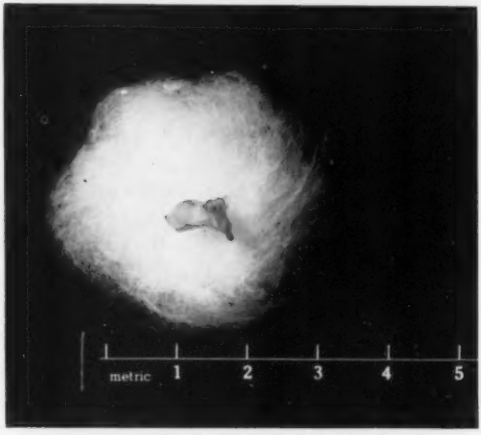

Fig. 4. Left arytenoid cartilage (Case 4).

If any arytenoidectomy or one of the cord displacement procedures is contemplated, due consideration should be given to the possibility of return of function of either or both vocal cords. A thorough clinical investigation, including basal metabolic studies and blood calcium determinations, should be carried out. The larynx and trachea must be carefully checked for evidence of cicatricial stenosis which may occur if a high tracheotomy has been performed.

EVALUATION OF THE VARIOUS OPERATIVE PROCEDURES.

The objective of all of the successful plastic procedures employed for the relief of bilateral abductor paralysis has

been to create sufficient glottic space (approximately 4 mm.) for adequate respiration, and at the same time to maintain the vocal cords sufficiently near the midline that they may be approximated by tension produced by the cricothyroid muscle and a serviceable voice may be sustained.

The King operation is primarily a cord displacement procedure utilizing the extralaryngeal approach. The Kelly operation, likewise, is an extralaryngeal procedure, but increased glottic space is accomplished by the removal of the arytenoid cartilage. Various modifications of both the King and Kelly operations have been proposed in an effort to facilitate dissection and render the procedures less formidable.

In order to simplify the procedure for arytenoidectomy, Thornell[22] proposed that the removal of the arytenoid be accomplished by an intralaryngeal technique utilizing the Lynch suspension apparatus. Prior to the advent of antibiotics, the intralaryngeal approach would have been obviated because of the likelihood of postoperative infection. Some laryngologists still are of the opinion that the extralaryngeal approach is preferable because of the possibility of cicatricial stenosis. In order to perform the Thornell operation, one must be proficient in the use of the Lynch suspension apparatus.

There has been some question as to the ability of a simple arytenoidectomy to accomplish satisfactory results. Wright[25] proposed a modification of the Kelly operation in which he sutured the posterior extremity of the vocal cord to the external perichondrium of the lower margin of the window in the thyroid lamina. More time will be required before a definite decision can be made as to whether Thornell's intralaryngeal operation will accomplish and maintain results that are comparable to the King and Kelly procedures. As regards the artenoidectomy by the laryngofissure technique described, there are certain advantages as well as disadvantages to be considered.

In any operative procedure, exposure of the field is of paramount importance. Certainly, by the midline thyrotomy inci-

sion, one is afforded an excellent view of the operative site as well as the surrounding laryngeal structures. Dissection, hemostasis, and primary closure of the laryngeal mucosa are all accomplished rather easily in a short period of time.

There are some who would disregard the midline thyrotomy approach from the outset because it is performed intralaryngeally, increasing the possibility of intralaryngeal infection. It must be admitted that time may prove the intralaryngeal methods to be unacceptable. Actually, there is slightly greater chance for exuberant granulation to form in the larynx by the laryngofissure technique because the anterior commissure of the larynx is divided in addition to the incision made in the aryepiglottic fold over the arytenoid.

Although the results of the four patients operated upon by this technique have been gratifying, a larger series of cases and considerably more time must elapse before any conclusions may be drawn as to the acceptability of the procedure.

A simple arytenoidectomy may, in some instances, fail to give adequate glottic space. Because it is very important to have a serviceable voice after the operation, the width of the arytenoid cartilage is about as much space as should be left. If the laryngeal airway is inadequate, a tracheotomy tube may be utilized; however, if after the operation a large glottic space has been afforded but the voice is inaudible or no better than a coarse whisper, there is little likelihood of improving the voice. It is for this reason that Negus[17] employs the valvular tracheotomy tube and does not risk impairment of the voice.

If the results of intralaryngeal arytenoidectomy prove comparable to those obtained through reposition and fixation of the vocal cord in the abducted position by the extralaryngeal method, the Thornell operation may evolve as the operation of choice; however, those who may hesitate to perform an arytenoidectomy through the Lynch suspension apparatus may feel justified in utilizing the laryngofissure technique.

SUMMARY.

When both recurrent laryngeal nerves are injured, as occasionally occurs during a thyroidectomy, a flaccid paralysis of all the intrinsic muscles of the larynx, except the cricothyroid, occurs, and the vocal cords assume a position at or near the midline. Although many theories have been advanced to explain the adduction of the vocal cords following the lower motor neuron paralysis, the exact cause remains to be proved.

Until 1939, no satisfactory method had been devised that would relieve the associated dyspnea and at the same time preserve the voice. Attempts to reunite the severed nerves or to anastomose them with other nerves failed to re-establish the function of the recurrent nerves. Early operative procedures were directed toward improving the laryngeal airway by removal of the obstructive vocal cord. Many of these operations were successful in alleviating the dyspnea, but the voice was markedly impaired; also, since these procedures were performed in the preantibiotic era, postoperative infection with resultant cicatricial stenosis was frequent.

Since King[12] devised the extralaryngeal cord-displacement operation in 1939, there has been a recrudescence of activity in operative procedures directed toward the correction of the disability produced by bilateral abductor paralysis. With the advent of chemotherapy, interest in the intralaryngeal approach has been renewed, and excellent results are now possible by both extra- and intralaryngeal surgery.

A modification of the intralaryngeal approach is presented in which the arytenoid cartilage is removed by the midline thyrotomy technique. Four patients have been satisfactorily rehabilitated by this method.

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TRIGEMINAL SYMPTOMS IN ACOUSTIC NERVE TUMORS.

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The symptoms and signs of acoustic nerve tumors frequently follow a relative pattern from onset to time of recognition and treatment, although the symptom pattern may vary in different individuals. Experimental and clinical studies have been in progress since the last decades of the 19th Century, but it was not until Cushing's[1] classic treatise in 1917 that diagnosis and treatment of acoustic tumors showed great improvement. As recently as 1949, Olivecrona[2] deplored the inability of the non-specialist to recognize acoustic nerve tumors in the early stages. Lundborg,[3] in review of 300 cases, stated that an otologist should be able to establish a preliminary diagnosis with routine methods in the majority of cases, if the entire symptomatology essential to the diagnosis is known. The most common early symptom is disturbance in hearing, for which the patient usually consults an otologist. According to Lundborg, the use of the Bekesy audiogram will greatly aid in the early diagnosis of acoustic tumors. Retrocochlear lesions show no recruitment or recovery of capacity to hear at higher levels of intensity with failure to hear at threshold intensity, whereas cochlear lesions show a recruitment. When there is a perceptive loss of hearing without recruitment, an acoustic tumor should be suspected.

In 300 cases analyzed by Lundborg, cochlear and vestibular symptoms were the first to make their appearance in approximately 71 per cent of the cases. Headaches were second in frequency in 16 per cent of the cases. Trigeminal symptoms were first noted in 6.3 per cent of the cases. Of

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these, anesthesia was noted in eight cases, paresthesia or hyperesthesia in six, and neuralgic pain in five. List,[4] in analyzing 175 cases, found trigeminal symptoms as the initial complaint in 6 per cent. Darquier and Schmite[5] noted trigeminal symptoms in 7 per cent of the cases among 43 analyzed. Edwards and Paterson[6] stated that trigeminal symptoms were present in 63 or 42 per cent of 175 cases and were initial in 16 or 10 per cent. Intermittent pain was present in four, numbness in 45, and paresthesias in 21. From the objective standpoint, they found trigeminal signs in 88 per cent of the patients or 138 cases. The corneal reflexes were diminished in 59 and absent in 78 or 50 per cent. Facial sensation was impaired in 62 per cent or 97 cases. In one, there was increased irritability to sensation of the first and second divisions. Pain and light touch were impaired in 76, pain in 13, and light touch in eight. Motor weakness was present in 31 and wasting was noted in five. Gonzalez Revilla[7] found that trigeminal symptoms were present in 56.5 per cent of 145 cases. These were of neuralgic form in 11 per cent, as compared to 7.5 per cent of Parker's[8] cases, 5 per cent of List's, and 1.6 per cent of those analyzed by Lundborg. This difference can perhaps be explained as a different interpretation of true neuralgic pain by various authors. Dandy's[9] experience and the finding of tumors as the causative factor for trigeminal neuralgia is well known. Objective trigeminal signs are present in the vast majority of patients with acoustic tumors: Parker 98 per cent, Edwards and Paterson 98 per cent, and the present series with 100 per cent. The most common sign is that of the impaired to absent corneal reflex. Diminished response to pain and temperature is next in frequency.

The physiology of trigeminal symptoms of acoustic nerve tumors is not difficult to understand. The acoustic nerve normally measures about 2 cms. in length, of which one-half lies in the posterior fossa, the other half being in the acoustic canal. The trigeminal nerve lies above the acoustic nerve and leaves the pons almost at a right angle to cross the petrous ridge; also, the descending tract of the fifth nerve is in relative proximity to the acoustic nerve at its pontine

junction. With these anatomic relations in mind, it is readily seen that a tumor of the acoustic nerve must of necessity compress both the posterior root of the fifth nerve and its descending tract when the tumor has reached medium size. The tumor need be no larger than a cherry to partially compress the trigeminal nerve, which seems to be particularly sensitive to extraneous pressure. On the other hand, the facial nerve is very resistant to pressure and may show little evidence of involvement even in the presence of a very large tumor. The vestibular nerve, as demonstrated by Schlittler,[10] is relatively resistant, but the cochlear nerve is vulnerable to compression. While the so-called trigeminal pain of a neuralgic type may be caused by acoustic nerve tumors, objective examination usually reveals evidence of sensory loss and particularly a loss of the corneal reflex. It seems apparent that acoustic nerve tumors may exist without clinical symptoms. Hardy and Crowe[11] found six acoustic nerve tumors in the internal auditory canal in 250 temporal bone dissections. It is possible that small tumors may occur in the nerve outside of the canal and compress the fifth nerve to produce irritative symptoms. Dandy[9] has noted the presence of tumors as the causative agent of trigeminal neuralgia in his discussion of the posterior approach in trigeminal neuralgia. The vestibular nerve and the facial nerve, being particularly resistant to compression, might in this case show no symptoms referable to these nerves. Since the trigeminal and the cochlear nerves are vulnerable, facial numbness or pain and hearing disturbances occur relatively early. In 35 patients seen with acoustic nerve tumors by the authors, objective trigeminal symptoms were present in all. They were known to be the initial symptoms in two and thought to be initial in a third. All of the patients, with the exception of one, had large tumors and presented no diagnostic difficulty when first seen in neurosurgical consultation. No tumor was smaller than an English walnut, and three were classified as being as large as a peach. The corneal reflex was diminished in all patients and known to be absent in 23. The area of greatest sensory loss was in the second division and in the area about the mouth extend-

ing well into the third division. The first division was least involved and, from the facts noted in the records, sensation was absent in four patients and diminished in six others. None of the patients had true neuralgic type of pain, although one had questionable sharp, shooting pains prior to the onset of numbness. Masseter weakness was recorded as being present in 12, while ten had very marked facial weakness. Masseter or temporal atrophy was not recorded in any of the cases. In only two of the cases was sensory loss a major complaint. Loss of hearing, disturbance in balance and co-ordination, clumsiness, and visual disturbances were predominate symptoms with the sensory symptoms playing a minor role in these patients with a large tumor. A third patient had a feeling of a frozen face for an indefinite period of time prior to the onset of hearing disturbance but could not remember whether or not he had any facial numbness.

Case 35 is of sufficient interest to warrant reporting in detail as an example of facial numbness being the major symptom in an acoustic nerve tumor and sufficiently disturbing to warrant surgical treatment.

Case 35: G. W., female, aged 62. This patient had been well until about two months prior to neurological consultation, when she began to note tightness and numbness of the right side of the face which slowly progressed to a complete right facial numbness. Immediately after the onset of the numbness, she noticed that if she would start or turn suddenly, she was unable to control her direction or stop her movement. For a moment or two she would stagger as though she had had one too many drinks. The complaint of staggering had not increased to any extent during the last month. She had noted that her hearing was poor in the right ear on one occasion but, following the removal of wax from the right ear canal, her hearing returned to what she considered normal. She had no headaches and no tinnitus of any type in the ear. Repeated questioning and suggestions could not bring out any complaints of abnormal sound in the ear. She stated that, other than the above complaints, she had never been sick and had not been under a doctor's care for many years and then only for the delivery of her children. The family history was entirely irrelevant. The patient's habits were good. She drank coffee but used no tobacco or alcohol and had lived a normal life as a housewife.

The examination revealed a well nourished, well developed white female of 62. There was no skull tenderness to percussion or deep palpation. The percussion note of the skull was normal; there was no bruit. Sense of smell was intact to peppermint, soap, and tobacco. The pupils were equal, regular, and reacted to light and accommodation and consensually. The visual fields were normal to rough tests. The visual acuity with

her glasses was normal. The optic fundi were entirely physiologic for the age. All extra-ocular movements were intact with no nystagmus or nystagmoid movements. There was complete right facial anesthesia except for a small border below the hair line on the right side of the face. Cotton touch was not recognized. Pin prick was recognized only as pressure. Cold and heat could not be differentiated. Masseter function on the right was slightly weaker than on the left. Both temporal muscles contracted about equally. Both sides of the face moved equally well on volition and emotion. Both platysmae contracted equally well and the palpebral fissures were equal. Hearing was questionably diminished to a C-256 tuning fork on the right, whereas a C-128 was about equally well heard in either ear. Watch tick was heard at two inches in the left and one inch in the right. An audiometer test was not performed. The palate moved in the midline on phonation. There was no falling away or past pointing of the outstretched extremities. The finger-to-nose, finger-to-finger, and heel-to-knee tests were well performed without ataxia. The gross and fine movements were fairly well performed and equally so on the two sides. All deep tendon reflexes were very active throughout but questionably more so on the left, particularly the left biceps being slightly more active than the right. There was no Hoffmann's thumb sign. The plantars were equivocal. The abdominals were present but very sluggish. Sensation was intact to all modalities. There was a slight unsteadiness in the Romberg position, but the patient did not grab for or hold onto anything in standing erect with her eyes closed and her toes and heels together. She did not turn to either side in the Babinski-Weil test.

The patient was hospitalized with the tentative diagnosis of a right angle tumor. The above findings being noted on neurosurgical examination, the diagnosis of possible Gasserian ganglion tumor, but probably angle tumor, was made. It was decided to explore the Gasserian ganglion first and then to explore the right angle if no tumor was seen in the ganglion.

Accordingly, a one-and-a-half inch incision was made extending upwards from the right preglenoid tubercle. The temporal fascia and muscle were divided in a straight line, and an opening the size of a quarter was made in the squamous portion of the temporal bone. The dura was elevated and the normal Gasserian ganglion was readily seen. The wound was then closed in the usual manner in anatomic layers, and a straight incision was made medial to the mastoid process on the right and an opening the size of a silver dollar was made in the suboccipital bone on the right side. The dura was opened as a flap, and a normal appearing cerebellum was seen. The cerebellum was gently elevated off the floor and we came immediately upon a soft, pedunculated tumor about the size of a cherry occupying the right angle with its tip lying against the pons. This was gently teased out, and the tumor extended as a stalk into the acoustic meatus. It was seen to be an acoustic nerve tumor, from which the VIIth nerve was easily separated. It was divided at the pons, and then an attempt was made to free the entire tumor from within the acoustic canal. This was impossible, as the tumor stalk broke off in trying to free it from the acoustic canal. It was then decided to sacrifice the VIIth nerve in order that the tumor be completely and permanently removed. Accordingly, the canal was curetted and a coagulation current applied within the canal.

The patient made an uneventful post-operative recovery but had a right facial paralysis and loss of hearing in the right ear. Two weeks later a hypoglossal-facial anastomosis was performed, following which

Dr. William Frackelton performed a corrective splinting with fascia lata strips. The facial numbness began to clear within a week after the operation, and within six weeks it was completely cleared except for the tongue, which has remained subjectively stiff. There has been a rather marked subjective ataxia or ataxic feeling with a moderate objective ataxia which is slowly but steadily improving. The face is beginning to move in somewhat of a mass form. It is expected the patient will make a very satisfactory recovery.

COMMENT.

This case represents one in which the major symptom of facial numbness became so acute and bothersome that surgical removal of a small acoustic neurofibroma was accomplished. If the facial numbness had not been so marked, it is probable that the tumor would have grown to a much larger size before causing other symptoms necessitating or bringing about surgical removal.

DISCUSSION.

A review of the literature and analysis of large groups of cases and a review of the authors' cases reveal that trigeminal symptoms are present in the majority. When the tumors are large, the trigeminal symptoms are pronounced because of the vulnerability of the posterior root of the Vth nerve and its descending tract to extraneous pressure. The presence of facial numbness and particularly corneal anesthesia, as demonstrated by loss of the corneal reflex in association with the symptoms referable to the VIIIth nerve, should immediately excite one's attention to the possibility and even probability of the existence of a relatively large tumor in the cerebellar pontine angle on the affected side. Otologic examination should seldom fail to establish the diagnosis in the presence of the above mentioned symptoms. Use of the recruitment test should be more generally understood and used to examine the function and status of the VIIIth nerve. The diagnosis and surgical treatment of acoustic nerve tumors in the relatively early stage will lead to a very high recovery rate with a minimum of residual symptoms. It is generally accepted among neurosurgeons that surgical treatment should be directed towards the total removal of the tumor. The VIIth nerve will, of necessity,

be sacrificed in a high percentage of these cases, but its repair can usually be accomplished with a relatively low disfiguring disability.

There is an increasing tendency to suspect organic pathology in the form of tumors or compression lesions in the presence of trigeminal symptoms of neuralgic form. Dandy[9] and, more recently, Sjoqvist[12] have emphasized this. On the other hand, Horrax and Buckley[13] lay little or no stress on fifth nerve symptoms in the differential diagnosis of acoustic nerve tumors. It would not be amiss for all patients with trigeminal neuralgia to have a very thorough otologic examination before the Vth nerve is partially or completely divided. The examination should not be limited to a mere evaluation of the patient's ability to hear a tuning fork but should also include the patient's capacity to hear at high and threshold levels of intensity.

It should be evident that combined efforts and cooperation between the otologist and neurosurgeon will bring about early recognition and treatment of acoustic nerve tumors. The neurosurgeon in particular should seek the otologist's aid more frequently in patients with trigeminal neuralgia, and the otologist should be alert towards acoustic nerve symptoms and have neurosurgical treatment early in the course of the lesion.

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PAN-AMERICAN MEDICAL WOMEN'S ALLIANCE, INC.

The IV Congress of the Pan American Medical Women's Alliance will be held at the Beekman Towers Hotel, First Avenue and 49th Street, New York, from September 24 through October 1, 1953. The morning hours will be given over to a scientific program of general interest. There will be round-table luncheon discussions with leaders speaking Spanish and English. Through the sponsorship of leading New York Medical Women those interested in a special field will have opportunities to attend clinics of their choice. The Medical Women's Association of New York is serving as hostess and planning a most delightful program. Hotel reservations should be made direct with the hotel. Registration fee is $10.00. Mail registration to: Registration Chairman: Ina A. Marsh, M. D., 140 Linwood Ave., Buffalo 9, New York.

LARYNGEAL FOREIGN BODIES WITH ONE CASE REPORT.*

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Although uncommon, the possible existence of a foreign body in the larynx must be considered, particularly in the young patient who suddenly develops one or more laryngeal symptoms such as aphonia, dysphonia, stridor, dyspnea, wheezing, cyanosis, and a croupy cough. The young patient in whom a history of aspiration is unobtainable is more prone to be misdiagnosed and treated for a longer period of time for other lower respiratory diseases. Out of a series of 50 cases in which foreign bodies were found in the air passages, Bonnier[1] found four laryngeal foreign bodies. In a 22-month-old child, a pericarp of an apple had been present in the larynx for two months. In a two-year-old child, a triangular piece of sheet iron had been present for 12 months. In both of these patients there was marked respiratory difficulty.

Withers[2] mentions various other reports of the presence in the larynx of a bone paper knife, a partial upper denture, a screw, a U-shaped "eye" of a "hook and eye," a metal rivet, as well as leeches, ascaris, trichina, and echinococcus. His own case, that of a 55-year-old seaman with a dime lodged in the horizontal plane of the larynx rather than in the usual and anatomically most suited vertical position, was unique.

In an analysis of 4,257 consecutive cases at the Chevalier Jackson Bronchoscopic Clinic,[3] an unlimited variety of foreign bodies was found in the air and food passages. The only limitations on variety were those of the individually invaded passages. The public as well as the medical profession owes

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its present knowledge to Jackson's great work in this field. The medical profession, however, is still not so alert or so aware of these conditions as it should be.

CASE REPORT.

A 14-month-old girl was referred to this hospital for hoarseness and audible breathing. The mother stated that the child had been well until three months before admission, at which time she suddenly developed a cough, hoarseness, and noisy breathing. The patient had been treated with antibiotics, cough syrups, and other remedies for "bronchitis." The cough subsided, but the voice change and noisy breathing persisted. Physical examination was normal except for a hoarse whispering voice and a soft stridor. There was no wheezing, tracheal slap, or other signs of a foreign body. Routine laboratory work and chest X-ray were normal. Under light ether anesthesia, direct laryngoscopy revealed a foreign body fixed in the glottic chink in the sagittal plane. The object had the appearance of a piece of septal cartilage. There was minimal swelling of the laryngeal structures. With the use of alligator forceps and by gentle tugging, the foreign body, a triangular piece of plastic from a child's toy (see Fig. 1), was easily removed. The hoarseness and stridor disappeared.

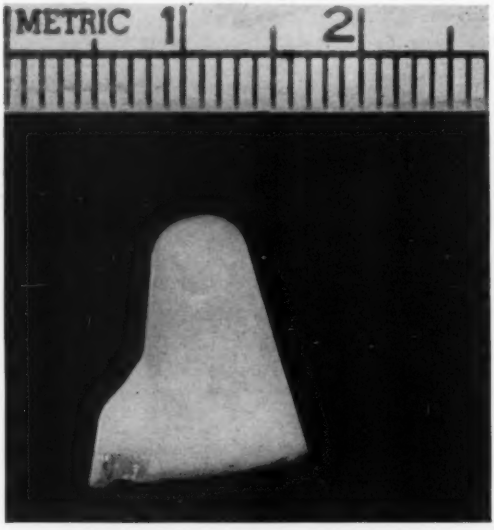

Fig. 1. Piece of child's plastic toy removed from larynx. Width measured about one-quarter cm.

Some authorities consider anesthesia unnecessary in such cases. Others feel that if no respiratory distress is present, a light ether anesthetic in the young patient is not dangerous and may facilitate the removal as well as cause less physical and psychological trauma.

SUMMARY.

The possibility of a foreign body in the larynx, particularly in the young patient with sudden or prolonged laryngeal symptoms, should be considered by all members of the medical profession.

The foreign bodies encountered in the air and food passages are many and greatly varied.

A case has been presented of a 14-month-old child in whose larynx was found part of a plastic toy, which had been present for three months.

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CORROSIVE ESOPHAGITIS DUE TO NITRIC ACID†

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The seriousness of the ingestion of a corrosive substance is never fully realized, even during the acute initial phase. Although the incidence of such accidents has decreased during the past decade, the average physician will still encounter an occasional case of corrosive esophagitis, either from accidental or suicidal ingestion. One fatal case of accidental concentrated nitric ingestion is reported to show the problems existing in the management and treatment of acid burns. In addition, an experimental study is presented to correlate the early pathologic findings with the therapeutic regime.

ETIOLOGY.

The most common causative agents of corrosive esophagitis are the alkalis. Much has been written concerning the pathology and treatment of caustic burns, but relatively little about the management of acid burns. Acid burns occur much less frequently than those caused by caustics. Holinger and Johnston[1] reported 96 cases, of which only five were produced by acids (sulphuric and nitric). In Vinson's[2] series, nine acid or acid-type burns were found in 186 cases.

A higher incidence of all types of esophageal burns is found in children below 10 years of age than in older individuals, due to carelessness by adults in the usage and handling of corrosive substances. Most of these accidents occur in the home, where the caustics are often thoughtlessly placed

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in a common household container such as a milk bottle, and within easy reach of children. The incidence in children is about equally divided between the two sexes. In the adult group, burns result not only from accident but also from suicidal intent, the latter cause being more frequent in this group. The group includes psychopaths, who swallow corrosive substances in a feeble suicide gesture, those who swallow a large quantity of the caustic during a severe depression cycle, and young women who attempt suicide for reasons of frustration or acute emotional disturbances.

Since cases of severe nitric acid burns of the gastro-intestinal tract present an unusually grave problem and few have been reported in the last 40 years, the following case report and study are presented.

REPORT OF A CASE.

W. W., a 56-year-old white male printing press operator, mistook a bottle containing nitric acid for one containing mouthwash on December 1, 1952. He gargled with 30 to 40 cc. of the concentrated acid; immediate gagging resulted, and he expectorated most of it, but swallowed an unknown amount. Upon discovering his mistake, he drank a quart of milk and called his family physician, who ordered him to a local hospital for treatment. His blood pressure was 120/60, temperature 99.8° F., pulse 104, respiration 24. An immediate blood count showed R.B.C. 4.95 million; W.B.C. 15,700; hemoglobin 14.8 gms. Other than a slight trace of albumen, the urinalysis showed no abnormality. Olive oil 2 ozs., a 5 percent solution of sodium bicarbonate, and bismuth subcarbonate, 2 gms., were administered every four hours. In addition, he was given intravenous fluids for shock, sedatives to control pain, and Atropine sulphate to help diminish salivation. An unsuccessful attempt was made to pass a Levine tube. On December 2, the patient was transferred to the Illinois Eye and Ear Infirmary for further treatment.

Physical examination revealed a pulse rate of 104, blood pressure 126/80, and a temperature of 101°. The patient was not in acute distress but showed a severe necrotizing cheilitis, stomatitis and pharyngitis. There were a few rhonchi bilaterally and a few moist rales at the base of the right lung. On deep palpation of the left epigastric area, a moderate degree of tenderness was noted, but there were no other significant findings. Penicillin, intravenous fluids and sedatives were administered and oral feedings and medication were discontinued. X-rays of the chest and neck (see Fig. 1) on December 3 disclosed soft tissue swelling in the hypopharynx and in the upper third of the esophagus. The left lung was clear and the right lung revealed some areas of old fibrosis at the right base. The heart and mediastinum appeared within normal limits. Since the patient was unable to swallow liquids or even his own saliva, on December 3, 1952, an attempt was made to pass a No. 30 Hurst mercury bougie, which encountered a complete obstruction at the entrance to the esophagus. The problem of feeding the patient was of considerable concern, and it was felt that a feeding tube

should be inserted, if possible. The following day, under topical anesthesia, an adult laryngoscope was carefully introduced. The walls of the hypopharynx, the epiglottis, arytenoids and pyriform sinus areas were visualized and found to be edematous and covered with a gray necrotic exudate. With care, a soft tip No. 12 bougie was passed through

Fig. 1. Lateral X-ray of neck, showing marked edema of epiglottis, arytenoid area, and cervical esophagus.

the cricopharyngeus stricture and esophagus with no appreciable resistance. Following removal of the bougie, a No. 14 plastic Levine tube was passed through the left nostril, and under the direct visualization afforded by the laryngoscope, the tube was slowly inserted without

resistance into the esophagus with the aid of a Matthew forceps. A chest X-ray showed the tube extending to the cardia, so for the next five days the patient was given tube feedings of clear liquids, with no apparent ill effects. His temperature continued to remain between 100° and 101° rectally.

On December 9, the eighth day following the accident, the patient coughed up a few necrotic plaques, 2 to 3 cm. in diameter, from his hypopharynx. Indirect mirror examination disclosed the tube to be in the left pyriform sinus, with no evidence of pressure on the arytenoids or cricoid area. On the morning of December 10, the ninth day following the accident, he began coughing and expelled the Levine tube. This was followed by the expectoration of a few plaques and small blood clots. A chest X-ray showed no change from previous chest films. While examining the hypopharynx with a laryngeal mirror, the patient began coughing and expelled a long tubular mold which appeared to be the complete inner lining of his esophagus. It measured approximately one foot in length, and was gray and necrotic in appearance. (See Figs. 2 and 3).

Fig. 2. Part of the expelled esophageal mold with a catheter in the lumen, and the proximal end open, showing thickness of the wall and necrotic mucosal structure. The wall varied in thickness, with some areas including layers of sloughed esophageal musculature.

Immediately following this episode, the patient displayed no evidence of shock, complained of no pain, and examination showed his blood pressure to be 110/68, pulse 88 and regular, and respiration 24. Auscultation of the chest revealed no evidence of widening of the mediastinum, and the lung fields were clear except for a few rales at each base. An immediate chest X-ray disclosed no change from the previous film. Intravenous fluids and a blood transfusion were administered, and a gastrostomy was considered; however, he began to vomit small amounts of blood and soon developed a state of shock, which necessitated repeated blood transfusions. His breathing became increasingly difficult and an emergency tracheotomy was performed. He continued to vomit blood and small amounts of necrotic tissue over the next few days, and expired on the 15th of December, the fourteenth day following accident, from a massive gastric hemorrhage.

A post-mortem examination showed the tongue and pharyngeal mucosa to be markedly congested and edematous with areas of shallow, crusted ulceration. The epiglottis was shaggy and edematous. The larynx,

trachea and bronchi contained a large amount of thick, red, mucoid material which welled out of the smaller bronchi on compression of the lungs. The mucous membranes of the larynx, trachea and bronchi were bright red in color and appeared moderately edematous. The posterior wall of the larynx exhibited a small, bluish-red, elevated area measuring 3 mm. in diameter. Externally, this corresponded to a penetrating ulcer of the esophagus at the level of the top of the cricoid

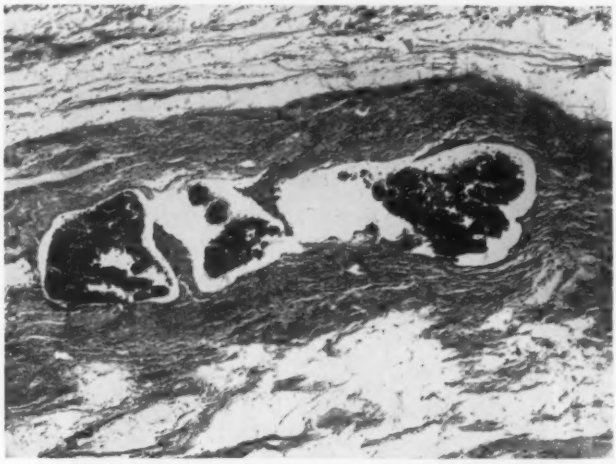

Fig. 3. Microscopic section of wall of the expelled cast of the esophagus revealing connective tissue infiltrated by numerous polymorphonuclear leukocytes. Many of the capillaries and veins contained recent thrombi. Note the marked degree of necrosis present throughout entire wall.

cartilage. A small perforation measuring 2 mm. in diameter was found in the posterior wall of the left main bronchus, 3 cm. distal to the carina; it communicated directly with the esophagus. The lungs were doughy in consistency and only in the upper lobes, was there a normal crepitant feeling to palpation. Under the surfaces of both lower lobes, there were many small, white, firm, flat nodules, the largest of which measured 1 cm. (See Fig. 4).

The esophagus was extremely congested and the walls were thin and friable. There was a thick, bloody exudate covering the entire wall and the mucosa appeared to be entirely absent, with muscle tissue exposed throughout the length of the esophagus. Small, flat, shallow ulcers were present over the entire surface; some penetrated into the muscular tissue of the esophageal wall, while several penetrated the entire thickness of the wall. One such penetrating ulcer extended into the retro-esophageal tissue over the body of the seventh cervical vertebra. Another penetrated the entire wall of the esophagus and larynx, except for the mucosa at the level of the cricoid cartilage. A third penetrated into the left main bronchus, as described previously,

and a fourth ulcer penetrated into the right side of the posterior mediastinum, where there was an abscess measuring 5 cm. filled with bloody, purulent material. The abscess showed no communication with the pleural cavity. The mediastinum was widened, especially in the posterior part just above the right diaphragm, where a soft, fluctuant, bluish area was visible and palpable.

Fig. 4. Postmortem specimen of larynx, esophagus and lungs viewed from the rear. Upper arrow shows 3 mm. superficial ulceration on posterior surface of the larynx. Lower arrow indicates a 2 mm. perforation in the esophageal wall, communicating by a fistulous tract with the left main bronchus.

The stomach was thin-walled, dark blue-gray in color, and distended. In the angle formed by the diaphragm, gastric cardia and the fundus, a 10 cm. abscess was located. It was filled with a thick, bloody, purulent material and communicated through a perforation with the stomach at the cardia. Upon opening the stomach, it was found to contain about 500 cc. of clotted blood and sloughed, dark-red, friable mucosa. There was an absence of the mucosa over the fundus and the proximal part of the antrum. The surface of the stomach was irregular with many shallow ulcers, especially in the cardia where the entire thickness of

the wall, except the serosa, was involved. On the serosal surface were yellow, friable plaques which were easily perforated. Over the proximal part of the antrum, the mucosa was very friable, deep red in color, and easily separated from the stomach wall. The remainder of the stomach was congested and edematous with a few small ulcers.

The duodenum was distended with blood clots. In the proximal part, the mucosa was markedly congested and edematous; the remainder of the gastro-intestinal tract contained a large amount of blood (estimated to be 1000 to 1500 cc's.) in various stages of digestion. The peritoneal cavity contained no fluid and the peritoneal surfaces were smooth and glistening; the heart, liver, spleen and kidneys showed no significant findings.

DISCUSSION.

A *diagnosis* of acid ingestion is usually determined from the history alone; however, the history may not be apparent in minimal cases, especially those of young children. All suspected cases of acid ingestion, even though history is vague, should be considered as corrosive esophagitis until proved otherwise. The degree of an esophageal burn cannot always be determined by the condition of the oral cavity. In some cases, the esophagus can be markedly constricted, yet the mucous membrane of the mouth or pharynx shows little or no involvement.[3]

Depending upon the amount and concentration of the acid ingested, the signs and symptoms of corrosive esophagitis will vary. In the moderate or severe case, the diagnosis is usually obvious from the history and physical findings. The patient complains of pain in the mouth, pharynx, and often in the left epigastric area. There is associated a profound thirst, fever and dysphagia, which vary in degree. The color of the mucous membranes of the lips, oral cavity and pharynx is usually gray, but may vary depending upon the acid ingested. A marked salivation, which is tenacious in character, is often present. When vomiting occurs, as happens frequently, the vomitus may or may not show evidence of blood. One difference in the findings of a severe acid burn as compared to caustic burns, is a greater degree of shock and toxicity. Gray[4] reported a case of a sailor who died from shock twelve hours following ingestion of nitric acid. A disturbance of the acid-base balance has caused death in other cases.

Dyspnea from laryngeal edema is not a common finding, but this condition should be kept in mind. Kearney[5] reported a case of sulphuric acid ingestion in which severe obstructive laryngeal edema occurred, necessitating an emergency tracheotomy.

In the early case, X-ray of the neck frequently discloses a marked edema of hypopharynx, epiglottis, and upper part of the cervical esophagus. It is generally believed that a chest X-ray shows no significant findings, but Beutel[6] believes that in the early period following ingestion of a corrosive substance, there is a change of the esophagus noticeable on X-ray. He states that acid produces a granulated outline to the esophagus without dilatation of the lumen, while alkalis cause the esophagus to be dilated and atonic.

Esophagoscopy in the early stages of a burn should never be performed as a diagnostic procedure. Little is to be gained and much may be lost. The only possible exception is in a case of an unsubstantiated history, with no evidence of mucosal findings in the mouth or pharynx. Even then, utmost caution must be observed.

An early estimation of the degree of stricture formation is generally a dangerous undertaking. If acid or alkali has been ingested, varying degrees of esophageal wall destruction are present. Obviously, healed scars do not form at once, although mucosal edema and ulceration will be present to cause severe obstruction. A barium swallow, or one of iodized oil, if there is clinical evidence of severe obstruction or perforation, will outline the areas of obstructing edema but will not necessarily indicate the sites of subsequent stenoses. Early esophagoscopy as a diagnostic visual aid in determining the degree of involvement is suggested by some, although this procedure is considered extremely likely to result in perforation because of the friability of the walls at this stage. For these reasons, while the history and findings indicate a corrosive esophagitis, the diagnosis of the presence of an esophageal stricture usually cannot be made for six weeks to two months following the accident. At this time, a barium swallow, or lipiodol if swallowing is associated with coughing,

will localize the strictured areas; however, multiple strictures may be present, interfering with the passage of barium and preventing a clear visualization of the lower areas. Esophagoscopy is then a confirmatory method for the diagnosis of a stricture, but is dangerous before two months, in spite of beginning scar formation histologically; however, here, too, in cases where there are multiple strictures, difficulty may be encountered in observing the lower ones.

The *pathology* produced by acids differs in some aspects from that of alkalis, although the resulting defects are similar. The pathology of esophageal burns may be discussed under two categories: acute and chronic phases. The extent of an acute burn depends on the type of acid, the amount, concentration, and duration of contact of ingested substance. Belinoff[7] uses three classifications to describe the amount of damage: (1) The light or mild are those cases with involvement of the mucosa and sub-mucosal layers by inflammation and edema. This group usually heals with little or no scarring or stricture formation. (2) The medium or moderately severe are those cases with involvement of the mucosa, submucosa and muscle layers. The affected layers show signs of inflammation, edema, and necrotic changes. It is common to have a sloughing of necrotic debris in small areas producing ulceration, or the whole esophageal lining may be expelled as an intact mold. The expulsion of the mold occurs between the eighth and tenth day. In the case herein reported, this happened on the ninth day. Others have also noted the expulsion of the esophageal mold. Cleland[8] reports a case in which the slough was expelled on the ninth day following the swallowing of hydrochloric acid. In the case reported by Lemariey and Billet,[9] this occurred on the tenth day, while Rubio and Martinez[10] reported the expulsion on the ninth day, after the swallowing of sulphuric acid in a 14-year-old child. Lee[11] describes a case of the esophagus being expelled on the ninth day following lye ingestion. Following the expelling of necrotic areas, scar formation begins through increased fibroblastic activity and collagen deposits. This can be noted as early as the third day following inges-

tion, and soft stricture formation may begin to develop by the second or third week, but during this interval, the patient may experience no difficulty in swallowing. (3) The grave or severe burns according to Belinoff's classification involve all layers of the esophageal wall, including serosa and periesophageal tissues. Perforations are common and complications frequently result. In a case reported by Ide[12], the esophageal wall was completely destroyed with the trachea resting on the vertebral column. The pathologic involvement is not necessarily limited to the esophagus in severe acid burns. The stomach frequently has a greater degree of mucosal damage than the esophagus because of the higher acid concentration. Trempe[13] reported the finding of a normal esophagus, but a markedly damaged gastric mucosa, following the swallowing of dilute nitric acid. It would appear that this is most unusual, since no similar cases appear in the literature. Death occurs in most severe cases either from immediate shock or toxic manifestations, or as a result of complications from perforations.

Although histologic evidence of the beginning formation of strictures may be noted as early as the second or third week, the development of chronic fibrous strictures may not be apparent until months or years after the accidental ingestion of small quantities of acid or alkali, when the patient begins to experience difficulty swallowing solid foods, or is treated for the removal of a foreign body in the esophagus. Complete atresia is not a rare occurrence in cases in which the developing stricture remains untreated. In the chronic case, the mucosa between strictures is thin and it is here that perforations usually occur. The site of stricture formation, in the order of frequency, as described by Jackson and Jackson,[14] is at the crossing of the left bronchus, the cricopharyngeus area, and at the diaphragmatic hiatus level. The majority of strictures in children are found in the upper part of the esophagus. It is believed that this is due to the small size of the lumen in this region which produces a temporary stasis in this area as fluids are swallowed.

The most frequent complications associated with severe acute esophagitis are mediastinitis, peri-esophagitis, pulmonary disease secondary to aspirated secretions, tracheo- or broncho-esophageal fistula, stomach perforations, and laryngeal obstruction from edema. Some of the complications in the chronic cases are anemia, prolonged starvation, avitaminosis, marked dehydration, and chronic pulmonary disease. Alsted[15] cited a case of pernicious anemia developing eight years following the ingestion of nitric acid. The prognosis in esophageal burns should be a guarded one, since the extent and exact localization of all pathology and the nature and severity of complications cannot be fully determined for several years.

EXPERIMENTAL STUDY.

In order to have a better understanding of the early pathology of acid burns, an experiment was conducted consisting of the introduction of nitric acid into the esophagus of an anesthetized dog, with subsequent study of the gross and microscopic pathology. The purpose of this study was to determine the condition of the mucosa of the dog's esophagus approximately 60 hours following the introduction of 5 cc. of concentrated nitric acid. The arbitrary period of 60 hours was chosen for two reasons: (1) In most cases, a two- to three-day period elapses before the otolaryngologist or esophagologist is consulted in regard to the patient; (2) Previous studies by Belinoff[7] indicate the greatest degree of damage to the esophageal wall is present at this time, and that collagen fibers begin to form the third day.

Method. A dog was anesthetized with intravenous barbiturates and placed in an upright position to achieve the desired physiological effects of uniform passage of the acid on swallowing, plus the effect of gravitation. An adult laryngoscope was introduced and the opening of the esophagus was identified. A pipette containing 5 cc. of concentrated nitric acid was introduced to the area of the inferior constrictor muscle. With the dog maintained in the vertical position, the contents of the pipette were allowed to flow freely into the esophagus, and as the solution passed through the con-

striction at the opening of the esophagus, the dog swallowed. He was maintained in this upright position until it was certain that the acid had passed into the stomach. A partial degree of sedation was administered by the Veterinary Department for the next 60 hours, during which interval a normal diet including liquids was maintained. Approximately 60 hours following ingestion of nitric acid, the dog was killed by means of an air embolism.

The esophagus and stomach were removed intact and examined grossly. The length of the esophagus was 29 cm. and examination of its mucosa disclosed varying degrees of necrosis along its entire course. The surface of the esophagus

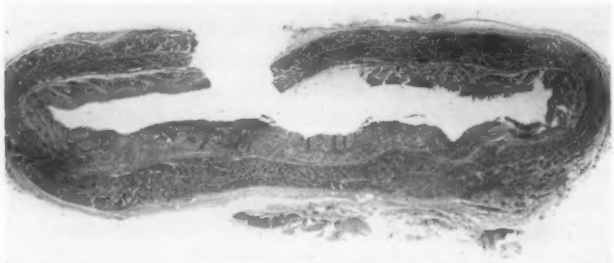

Fig. 5. Low power cross-section of dog's esophagus, revealing almost complete destruction of the stratified squamous epithelium. Only small islands of epithelium were preserved. The submucosa and the muscularis were infiltrated by numerous polymorphonuclear leukocytes. The muscular fibers were fragmented in various areas.

was a reddish-brown color with superficial shreds of friable tissue. A small ulcer was noted on the posterior wall of the esophagus 7 cm. below the constriction of the inferior constrictor muscle. It was $2\frac{1}{2}$ by 1 cm. in size, with muscle fibers forming the floor. At a distance of 15 cm., a linear ulceration, 8 cm. by .5 cm., was noted along the right posterior wall of the esophagus. This ulcer was more superficial and involved only the mucosal and sub-mucosal layers. In the region of the cardia, the mucosal surface was very dark brown and a circular ulceration 2 cm. in diameter was noted to penetrate into the muscle substance of the esophagus (see Figs. 5 and 6).

Examination of the mucosa of the stomach showed superficial necrosis over a large part of the greater curvature, with a few pin-point ulcerations. No areas of perforation of the stomach were observed. The mucosa in the region of the pylorus was normal in appearance.

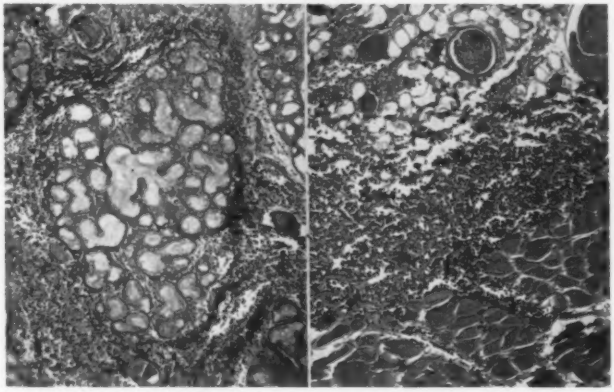

Fig. 6. The left illustration is a highpower section of Fig. 5, showing partial destruction of the mucous gland with marked polymorphonuclear leukocytic infiltration surrounding and between glands. At right is a highpower section of Fig. 5, showing abscess formation and polymorphonuclear leukocytic infiltration into muscle substance.

Microscopic examination of the esophageal wall disclosed almost complete necrosis of mucosa, sub-mucosa and mucosal glands. Several areas of leukocytic infiltration were noted with small abscess formations. In two instances, the abscesses were noted to have practically penetrated the muscle wall. Examination of the esophagus showed a partial necrosis of the muscle wall with marked inflammatory reaction. No perforation was observed in this area.

TREATMENT.

Prophylaxis should be considered first in a discussion of the treatment of acid burns. Many cases would not occur were it not for carelessness in the handling or storing of corrosive solutions.

First and foremost in the treatment of acute burns is the consideration of the patient as a whole, before initiating specific therapy. Often shock, acute blood loss, marked dehydration or acidosis are the first steps to be corrected before continuation of treatment. Once these factors have been corrected, it is imperative that treatment and dilatation be started early. This is particularly true since once the esophagus is burned, strictures can be formed at any time in the life of the patient.

The actual management of all acid burns should be approached in the same manner, since the degree of severity is not usually known. Generally, the first step is to give a neutralizing substance such as sodium bicarbonate, magnesium oxide or bismuth subnitrate. The use of small quantities of dilute alkalis at frequent intervals for the first five days seems indicated. After five days, the necrotic membrane would make this ineffectual; however, some, including Quist-Hanssen,[16] feel that neutralizing agents have no effect on the corrosion or general condition of the patient. Sedatives for pain, intravenous fluids for control of shock, and mild antiseptics for cleansing the mouth should be given. Antibiotics and chemotherapy should be given to lessen secondary infection or chances of complication. Krey[17] found in his experimental work that antibiotics and chemotherapy had no effect on the prevention of stricture formation, but were of value in helping control secondary infection. Olive oil can be applied to any burns on the lips. Gastric lavage should be attempted only if the tube can be introduced easily, because of the dangers of perforation or stimulation of the vomiting reflex.[18] The copious quantities of saliva should be aspirated. An early jejunostomy may be indicated in some cases.

Several methods have been advocated to help maintain a patent esophageal lumen and prevent the formation of strictures. In an acute case, some advocate inserting through the nose a Levine tube, which is passed through the esophagus into the stomach. This serves a dual purpose in providing means of supplying nourishment for the patient, and in helping maintain a patent lumen. The tube should not be per-

mitted to cause pressure on the larynx or cricoid cartilage area, or ulceration may result. If a Levine tube cannot be inserted, every effort should be made to have the patient swallow a string. This will at least maintain a pathway from the mouth to the stomach for future corrective procedures.

One of the more common methods used for prevention of strictures is early bouginage. There are two schools of thought as to the time such treatment should be started. Saizer[19] and Bokay,[20] believed in the passage of soft rubber bougies, beginning between the second and sixth day. In this method, active dilatation is begun within the first 24 to 48 hours following ingestion of the corrosive. Various sizes are used, depending upon the age of the patient. The bougie, passed by mouth, is carried by its own weight into the stomach. This procedure is performed daily for about two weeks and each time, if possible, a larger bougie is used. After two weeks, the time, interval can be increased, depending upon the clinical response of the patient. This technique has given excellent results and has completely changed the management of acute corrosive burns of the esophagus in many parts of the world; however, others maintain dilatations should not be started before ten days to two weeks because of the danger of perforation and of producing inflammation. Belinoff's[7] technique is bouginage between the seventh and tenth day, preceded by early diagnostic esophagoscopy. In certain cases, Jackson and Jackson[14] suggest bouginage through an esophagoscope under direct visualization from the second week on. Another method of treatment suggested by Plummer[21], and advocated by Vinson[22], Moersch and others, is the immediate swallowing of a thread, which is used as a guide at a later date for threading olive-tipped bougies for subsequent dilatation. Tucker[23] advocates retrograde dilatation; the patient swallows a thread, an early gastrostomy is performed, and a rubber bougie is drawn upward through the gastrostomy opening into position in the esophagus and allowed to remain for short periods of time to act as a dilator. This method is useful especially when multiple, fully formed, fibrous strictures are present.

A much more efficient technique which has proven to be safe and permits a more rapid dilatation of tight strictures has been suggested by Barretto,[24], Botelho[25] and Da Rocha, Da Cunha and Guilmaraes.[26] This combines several previously reported procedures. It is used in patients who have a gastrostomy opening and a continuous string, and consists of passing Plummer bougies along the string from above downward, the string being grasped and fixed firmly at the gastrostomy opening. This technique is continued until an esophagoscope can be passed the length of the esophagus with ease, at which time the string is removed, the gastrostomy allowed to close, and subsequent dilatations made with mercury-filled bougies or the esophagoscope.

In cases of complete atresia, it may be necessary to perform esophagoscopic bouginage from above and from below through the gastrostomy opening. A biplane fluoroscope is usually necessary in carrying out this procedure. Where bouginage is impossible in cases of complete atresia, it may be necessary to reconstruct an esophagus by surgical means. One of the technics used is the creation of subcutaneous skin flaps, forming a new esophagus. Another method is the resection of the atresia with an anastomosis of the stomach or jejunum to the upper part of the esophagus; however, the increasing frequency with which post-surgical strictures are being encountered makes one question the efficiency of these procedures, since these strictures require the same type of dilatation that was being performed for the caustic stricture itself.

At this point, the external esophagotomy as suggested by Belinoff,[27] and later Leegaard.[28] should be mentioned as a means of treatment for a severe esophageal burn; however, this procedure has not been widely accepted.

A complication frequently associated with severe esophageal burns is extensive damage of the mucosa of the stomach. This is especially true in cases of acid burns as indicated in this case report. When this condition occurs, a gastrostomy opening may be contraindicated, necessitating a jejunosto-

my. This operation is suggested as a routine procedure in cases of severe acid burns, instead of attempting a gastrostomy, in view of the damage to the stomach, as apparent in this case and from a review of the literature. Jejunostomy feedings can maintain nutrition, although this requires special feeding techniques.

SUMMARY.

1. Acid burns of the esophagus are much less common than alkaline caustic burns in the ratio of about one to twenty.

2. Acid burns appear to be much more severe, causing extensive erosion and necrosis of the esophageal and gastric walls, often complicated by the expulsion of a mold of the entire esophagus.

3. A fatal case of concentrated nitric acid ingestion is reported, in which the walls of the esophagus and stomach were perforated by the acid, and in which a mold consisting of the esophageal wall was extruded on the ninth day.

4. An experimental study of the effects of concentrated nitric acid on the esophageal wall is presented as a basis for suggested therapy.

5. Jejunostomy rather than gastrostomy is suggested in cases of acid burns.

6. Techniques of treatment of acid burns of the esophagus are reviewed. Early mercury-filled bougie dilatation is advocated except in severe acid burns, where such treatment appears to be contraindicated.

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GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will hold its Spring meeting March 5th and 6th, 1954 at the General Oglethorpe Hotel, Savannah, Georgia. The speakers will be Dr. Paul Chandler, Boston; Dr. A. B. Reese, New York; Dr. Henry P. Wagener, Rochester; Dr. L. R. Boies, Minneapolis; Dr. Francis LeJeune, New Orleans, and Dr. J. H. Maxwell, Ann Arbor.

TONSILLECTOMY AND POLIOMYELITIS*

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The 1952 outbreak of poliomyelitis in North America had a record-breaking incidence with an estimated 55,000 cases being reported in the United States alone (Van Riper[1]). The Saskatchewan epidemic was the worst ever experienced in this Province with an extremely high percentage of patients having respiratory involvements. According to the statistics released by the Provincial Department of Public Health (Dr. Kinneard[2]) there were 1,223 cases with 455 of paralytic type and 89 deaths. The peak of morbidity and mortality was in August and September — the months of "tonsillectomy epidemic".

The old tonsillectomy-poliomyelitis controversy rose again. A survey of available literature is necessary in order to present the various aspects of the tonsillectomy-poliomyelitis relationship. Here are the more important questions that come to light:

1. Is the part played by tonsillectomy such as to suggest that this operation increases the risk of contracting poliomyelitis?

As in other problems there is no unanimity. Anderson, Anderson, Skaar and Sandler[3] state that the risk is three times as great among recently tonsillectomized patients as among non-tonsillectomized. In Fisher, Stillerman and Marks'[4] series there was a higher incidence of poliomyelitis in the recently tonsillectomized children than in the general child population. J. A. Anderson[5] found that the incidence was 2.6 times higher among tonsillectomized children. In Britain, poliomyelitis cases following recent tonsillectomy were fewer

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than might be expected from United States experiences (Tamplin[6]). Aycock and Luther[7] feel that there is a causal relationship between tonsillectomy and poliomyelitis since the interval between the operation and the onset of polio corresponds to the incubation period of the disease; however this causal relationship exists only in rare instances.

Seydell[8] believes that this relationship cannot be determined without further extensive study. Page's[9] survey showed only one case of poliomyelitis among 8,915 recently tonsillectomized patients. Boies[10] is not convinced that tonsillectomy is a factor in the pathogenesis of poliomyelitis. In the discussion on Boies'[10] paper, Shurly[11] said that he had never seen such a relationship in more than 20,000 tonsillectomies; also McFarlan[12] in his studies did not record even one case of poliomyelitis with a history of a recent tonsillectomy — this may be due, however, to suspension of the operation during the epidemic. Pederson[13] found that the risk of poliomyelitis consequent to recent tonsillectomy is not considerably higher than the incidence of poliomyelitis in the general population during an epidemic.

Interesting statistical data is presented by Roberts[14] which prove that the annual incidence of poliomyelitis in the general population is thirty times greater than that in the recently tonsillectomized individuals during an epidemic.

Kinney[15] believes that the tonsillectomy-poliomyelitis relationship is not more than casual and that hysteria of the public is unwarranted. An elaborated formula has shown that the number of children with poliomyelitis after recent tonsillectomy was less than the normal expectancy. Cunning[16] who surveyed 35,039 tonsillectomies does not see any causal relationship between poliomyelitis and tonsillectomy. There is no proof yet, that children tonsillectomized during summer are more liable to contract poliomyelitis than the general child population. The alarm of the public and the medical profession is unfounded. Table 1 presents the comparison of the total polio cases to the number following tonsillectomy.

TABLE 1

TOTAL NUMBER OF POLIOMYELITIS AND NUMBER OF CASES FOLLOWING TONSILLECTOMY WITH TYPES OF INVOLVEMENT.

Author	Total Polio Cases	Total Polio Cases Following Tonsillectomy	Bulbar Type Following Tonsillectomy	Spinal Type Following Tonsillectomy	Mild or Unknown Type Following Tonsillectomy
Howard, Cincinnati Hospitals 1937-1943	237	6	3	3	—
Helms, 1937-1938	711	7	5	1	1
Pederson, 1941-1945	492	11	6	4	1
Cunning, 1946	2,290	56	18	24	14
Cunning, 1947	4,331	24	6	10	8
Cunning, 1948	10,624	39	27	8	4
Personal Communications. Yorkton General Hospital 1950-1952.	71	2	2	—	—

2. Does recent tonsillectomy increase the danger of developing bulbar type of poliomyelitis?

There is no general agreement that it does, and the statistics differ markedly. Anderson's[5] survey reveals a definite hazard of bulbar poliomyelitis following tonsillectomy performed during an epidemic; the incidence of bulbar and bulbo-spinal infection was found to be 16 times greater in recently tonsillectomized children than in the general child population. In Anderson, Anderson, Skaar and Sandler's[3] series—eleven times greater. Zellweger[17] cited 128 cases of poliomyelitis shortly after tonsillectomy with 68% of bulbar involvement. In Seydell's[8] survey of statistics the total number of cases in which the bulbar form followed a recent tonsillectomy was twice as much as the number of the spinal type. The prevalence of bulbar infection after tonsillectomy is obvious

in Eley and Flake's[18] study; there were 2.78% of spinal type and 12.98% of bulbar type of poliomyelitis following recent tonsillectomy, or a ratio of 5 to 1.

Aycock and Luther[7] expressed their view that there is a definite relationship between tonsillectomy and poliomyelitis in that an excessive number of cases of bulbar infection occurred at the same interval after the operation; however, Aycock[20] feels that the danger of bulbar poliomyelitis consequent to tonsillectomy "most of the time in most places is almost nil, but for short periods of time in some places during an epidemic it is a risk to be avoided." Pederson[13] states that when poliomyelitis occurs following tonsillectomy it is more apt to be bulbar in type.

Cunning,[21] having completed his four years' study of 36,678 cases of poliomyelitis and 96,379 cases of tonsillectomies did not see any conclusive proof that tonsillectomy predisposes to bulbar infection. He cites 1,481 bulbar cases of which 1,450 or 97.9% occurred without history of recent operations. Kinney[15] contends that the suggestion that the T. and A. operation during an epidemic increases the danger of the child's developing bulbar poliomyelitis belongs to the past.

Finally Mosher[22] in the discussion on Seydell's[8] paper said that from the clinical and experimental evidence no definite conclusions can be drawn as to the relationship between recent tonsillectomy and bulbar poliomyelitis, and that it is not yet proved whether a recent tonsillectomy may determine the type of polio infection.

The incidence of poliomyelitis following tonsillectomy with bulbar type of infection and deaths as reported by different authors are presented in Table 2.

On the total of 352 instances of poliomyelitis following recent tonsillectomy there were 183 cases of bulbar infection or over 50 per cent.

TABLE 2

POLIOMYELITIS FOLLOWING TONSILLECTOMY WITHIN 30 DAYS AND 30 TO 60 DAYS AFTER THE OPERATION

Author	Year	Total Cases	Bulbar Type	Number of Deaths When Stated
Shepard	1910	1	1	
Aycock and Luther	1929	16	7	
Ayer	1929	9	9	
Silverman	1931	5	4	
Pamment	1933	1	1	1
Stillerman and Fisher	1935-1937	13	9	3
Eley and Flake	1937	25	17	3
Fisher, Stillerman and Marks	1937	12	7	
Helms	1937-1938	7	3	No deaths
Howard	1937-1943	6	3	
Koskoff, Amshel and Lebeau	1938	2	2	2
Seydell	1940	20	15	
Krill and Toomey	1941	5	5	3
Zellweger	1941	40	6	
Pederson	1941-1945	11	6	
Anderson	1943	17	17	
Page	1944	1	—	
Lucchesi and La Boccetta	1944	3	2	
Roberts	1946	6	—	
Anderson, Anderson, Skaar and Sandler	1946	21	12	3
Kinney	1946-1947	5	1	
Cunning	1947	24	6	
Cunning	1948	38	27	
Cunning	1949	62	21	
Personal Communications	1952	2	2	2
TOTAL		352	183	

The records of the Yorkton General Hospital for the last three years (1950, 1951 and 1952) have been examined and the figures for tonsillectomies and poliomyelitis are presented in Table 3.

TABLE 3

TONSILLECTOMY AND POLIOMYELITIS IN YORKTON GENERAL HOSPITAL.

YEAR	1950			1951			1952		
Month	Number of Tonsillectomies	Total Number of Poliomyelitis	Number of Poliomyelitis Following Tonsillectomy	Number of Tonsillectomies	Total Number of Poliomyelitis	Number of Poliomyelitis Following Tonsillectomy	Number of Tonsillectomies	Total Number of Poliomyelitis	Number of Poliomyelitis Following Tonsillectomy
January	7	0	0	21	0	0	12	0	0
February	14	0	0	6	0	0	8	0	0
March	20	0	0	10	0	0	20	0	0
April	21	0	0	22	0	0	37	0	0
May	17	0	0	15	0	0	27	0	0
June	60	0	0	29	1	0	45	0	0
July	100	0	0	88	1	0	66	5	0
August	97	0	0	82	0	0	10	19	1
September	46	0	0	17	0	0	0	16	0
October	32	0	0	23	0	0	14	15	0
November	15	0	0	12	0	0	29	13 & 1	1
December	14	0	0	18	0	0	29	0	0
Total	443	0	0	343	2	0	297	69	2

It appears from these statistics that: *1.* There was no one case of Poliomyelitis in 1950. *2.* At the height of Tonsillectomies, in June, July and August 1951 only two cases of Poliomyelitis occurred and were not related to a recent Tonsillectomy. *3.* In September 1952 when tonsillectomies ceased completely in our hospital there were 16 admissions for Poliomyelitis. *4.* On the total of 69 Poliomyelitis patients in 1953 only in two was Tonsillectomy recently performed.

Here briefly are their histories:

Case Number 1. Hospital Number 1560 and 2621—52. F. T. 8 year old boy underwent T. and A. operation on May 17, 1952, and was discharged in fair condition May 19. Admitted again to the hospital on August 8, 1952, with typical symptoms of acute anterior poliomyelitis with bilateral paralysis of lower limbs including the waist. Spinal fluid: slightly cloudy; cell count—W.B.C. 285, polys. 90%, lymph. 10%, R.B.C. 42, gobulin markedly increased. After 3 days transferred to the Polio Clinic in Regina where the patient died of bulbar paralysis with symptoms of infection of higher centres.

It appears from the epidemiological investigation that the family was all well and carried on their usual normal activities at home until a sister recovering from poliomyelitis came from another town to stay with her family. One of her brothers who had not undergone tonsillectomy developed spinal poliomyelitis. The other brother who underwent tonsillectomy 11 weeks ago also developed poliomyelitis but of bulbar type. The first brother recovered from poliomyelitis; the tonsillectomized, died.

There was no epidemic of poliomyelitis at the time of the operation. The other feature of the case is fulminant spread of the disease in the tonsillectomized child who started with spinal infection and developed the bulbar type. We may assume that the recent tonsillectomy and abscence of tonsils influenced the course of infection in the tonsillectomized patient, other conditions (source of infection) being equal for both brothers.

Case 2. Hospital Number 3621, 1952. T. D., 3 year 9 months old boy underwent T. and A. operation on November 8, 1952, and was discharged in good condition on November 4, 1952. Seven days later the boy became ill and was admitted to the hospital of his home town. A spinal puncture was performed with the following results; W.B.C. 32 per cmm., erythrocytes 2 per cmm., faint increase in globulin, spinal fluid clear.

Epidemiologic investigation revealed that there was no epidemic of poliomyelitis in the home town of the patient. Six weeks prior to his illness there was only one case of poliomyelitis noted in that town, but on the same day of admission to the hospital of the tonsillectomized child, another boy was admitted with bulbar poliomyelitis. Both children were flown to Regina Poliomyelitis Clinic. The tonsillectomized patient

died within four days from bulbar paralysis; the other boy, who has not been operated on for tonsils did not die. He is still in the Clinic making good progress toward recovery.

We may presume that the patient contracted poliomyelitis in his home town, since at the time of his tonsillectomy there was no one case of that disease in our city. Once again the comparison of the course of the disease in both boys leads one to think that tonsillectomy had influenced the fatal outcome in one of them.

3. Does the presence or absence of the tonsils and adenoids influence the susceptibility to poliomyelitis, the type and the mortality of the disease?

Shepard[23] as long ago as in 1910 suggested that the tonsils and adenoids may offer a resistance to the virus invasion and, therefore, did not advocate tonsillectomy during an epidemic.

Fisher, Stillerman and Marks[4] found in their survey that the percentage of bulbar infection among the children who had a history of tonsillectomy, regardless of when performed, was twice as high as that among children with tonsils intact, who contracted poliomyelitis. They consider the tonsils to be a barrier against the virus.

Pederson's[13] figures proved that there is a higher incidence of bulbar and bulbo-spinal type of poliomyelitis in tonsillectomized individuals than in non-tonsillectomized, the ratio being two to one. The influence on mortality rate was demonstrated in a group of patients with tonsils intact as having six deaths from poliomyelitis, and in another group with tonsils removed as having 11 deaths.

The figures of Eley and Flake[18] are even greater; the bulbar form developed in 46.9 percent of the children with poliomyelitis whose tonsils have been removed in the past, as compared with only 17.9 percent of children with tonsils intact. In McFarlan's[12] survey the figures are 26 percent and 8 percent respectively. Lucchesi and La Boccetta[25] feel that although

the absence of tonsils and the occurrence of bulbar poliomyelitis may be coincidental, nevertheless, there is a definite relationship between the absence of tonsils and adenoids and the involvement of the higher centers in poliomyelitis. In their series the incidence of bulbar and bulbo-spinal poliomyelitis was considerably higher in patients without than in patients with tonsils and adenoids. They consider the Waldeyer ring as a protective barrier against the poliomyelitis virus, and oppose the indiscriminate T. and A. operation.

Top and Vaughan[26] are convinced that the tonsils have a definite influence on the infectious process in poliomyelitis. Aycock's[7] survey showed that the absence of tonsils favored the occurrence of bulbar type of the disease. Other authors do not share that view. Zellweger[17] believes that a previous tonsillectomy has no influence on the general morbidity of poliomyelitis. Tamplin[9] concludes that the incidence of poliomyelitis in tonsillectomized individuals is no greater than in those with tonsils intact. According to Aycock and Luther[19] the percentage of cases of poliomyelitis with recent tonsillectomy is so near, that of the general child population that the presence or absence of tonsils does not seem to influence the occurrence and course of poliomyelitis.

It is interesting to note that Vaughan, Vaughan and Palmer[27] found that in the New York epidemic in 1916, when 8,928 cases of poliomyelitis were reported with 2,407 deaths, the disease was rare among tonsillectomized children, and poliomyelitis affected mostly children with hypertrophied tonsils and adenoids.

The previous suggestions that a tonsillectomized child is more susceptible to poliomyelitis and that this susceptibility lasts for many years is criticized by Kinney[16], who says: "Nothing could be further from the truth."

Table 4 presents a comparison of poliomyelitis morbidity in tonsillectomized and in non-tonsillectomized children.

TABLE 4

POLIOMYELITIS MORBIDITY IN TONSILLECTOMIZED AND NON-TONSILLECTOMIZED CHILDREN

Authors	Number of Tonsillectomized Children	Total Polio Cases	Attack Rate	Bulbar Type	Attack Rate	Number of Non-Tonsillectomized Children	Total Polio Cases	Attack Rate	Bulbar Type	Attack Rate
Anderson, Anderson, Skaar and Sandler	2,686	16	0.6%	12	0.45%	240,779	491	0.20%	96	0.04%
Fisher, Stillerman and Marks	1,714	8	0.4%	4	0.2%	337,045	494	0.11%	59	0.018%
Pederson	?	148	***	12	***	?	206	***	5	****
Seydell, Wichita Epidemic, 1940	862	none	***	***	****	?	45	****	**	****
Tamplin, Portsmouth Experience, 1947	1,000	none	***	***	****	*****	***	****	**	****
Anderson	4,199	17	0.404%	17	0.404%	156,000	232	0.14%	39	0.025%

The striking features of Table 4 are: *1.* The higher incidence of poliomyelitis and its bulbar type in the tonsillectomized child population in most of the statistics. *2.* No one instance of poliomyelitis among 1862 recent tonsillectomized children operated on when when poliomyelitis was present in their communities.

4. Should the discontinuation of the T. and A. operation be recommended in the summer months? What time then would be the proper one? Is tonsillectomy an elective operation which can be postponed indefinitely?

The Boards of Health throughout the United States advise the suspension of any operations on the nose and throat during months and years of poliomyelitis epidemics (Howard[26]).

The majority of writers advocate postponement of T. and A. operations during the season of poliomyelitis especially in areas and communities where there is a high incidence of the disease (Fisher, Stillerman and Marks,[4] Helms,[36] Lucchesi and La Boccetta[25], Sabin[29], Evans [31], and many others).

As the problem of tonsillectomy-poliomyelitis relationship has been already raised, is wise, according to Mosher[22], to suspend T. and A. operation when poliomyelitis is prevalent.

Wesselhoeft[39] recommends that until we have a quick method of discovering the presence of the poliomyelitis virus in patients scheduled for T. and A. we must rely upon epidemiological data compiled by the Department of Public Health. We can carry on with the operation throughout the summer and fall providing there were no cases of poliomyelitis in the community.

Aycock[20] sees the best time for tonsillectomy to be changed from late summer "before school opens, to early summer" after school closes" but before poliomyelitis starts. Koskoff, Amshel and Lebeau[33] believe that if there is a seasonal rise of poliomyelitis in May or June epidemic proportions may be expected in August and September. The surgeon should watch the incidence of poliomyelitis in early summer to postpone eventually the T. and A. operation in late fall. Kinney[15] sees the solution of the problem in concentrating tonsillectomies and adenoidectomies in the months of April, May and June.

To postpone tonsillectomy in summer and operate later on means to increase the risk of upper respiratory infection. Cunning[34] feels that up until now we have no proper justification for doing so as no definite proof was produced in favor

of discontinuing T. and A. operation during the months of poliomyelitis epidemic and because our fear of poliomyelitis after tonsillectomy is unwarranted. The surgeon himself should decide whether the dangers of delaying tonsillectomy are greater than the risk of contracting poliomyelitis. Cunning[21] does not agree that tonsillectomy is an elective operation which can be postponed indefinitely for months. He considers tonsillectomy to be a preventive operation against nephritis, otitis media, rheumatic fever and other focal infections, and its delay may bring harm to the patient. Tamplin[6] feels the same way when saying "there is no doubt that if we unnecessarily postpone operating on tonsils and adenoids great harm, needless suffering, and serious ill health will result to hundreds of children who are in sore need of our assistance."

5. Can a simple answer be given to the problem of polio-tonsillectomy relationship?

We should say, no. Although the statistics presented are useful no definite conclusions can be drawn due to the striking differences in results obtained by various authors. These differences may be due to various strains of the virus involved on one hand and to the susceptibility and the resistance of the examined patient on the other.

The etiology and epidemiology of poliomyelitis is still subject to intensive investigation, and there is, as yet, no definite proof pointing to the route of transmission of the virus from the portal of entry to the central nervous system. The results of the experimental work are also inconclusive as it is not known how closely we can relate the findings in monkeys to human beings.

Up to now opinion varies markedly: some writers believe that there is no tonsillectomy-poliomyelitis relationship whatsoever; some are convinced that this operation should be banned when poliomyelitis is prevalent. It has been also thought that the surgeon being himself a healthy carrier infected his patients during T. and A. operation. Some others do believe that tonsillectomy must occur within the incubation

period of poliomyelitis in order to influence the course of the disease. In all probability tonsillectomy may precipitate poliomyelitis in children who are healthy carriers before the operation. As it is not known exactly what constitutes the susceptibility and the resistance of the healthy carriers to poliomyelitis it is wise not to operate in time of increased exposure to the virus. The education of the public in respect to the danger of poliomyelitis after tonsillectomy may also influence the decision of the surgeon.

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5 Dunlop Block.

THE AMERICAN OTORHINOLOGIC PLASTIC SURGERY SOCIETY, INC.

Meeting, October 11, 1953—Morrison Hotel, Chicago

PROGRAM

Board of Directors Meeting.....5:00 P. M.

General Membership Meeting.....5:30 P. M.

Cocktails .....6:30 P. M.

Dinner .....7:00 P. M.

Scientific Program .....8:00 P. M.

1. OPERATION—FACE LIFT.....By Dr. John F. Pick.
2. POSSIBILITIES AND LIMITATIONS IN PLASTIC SURGERY OF THE NOSE.....By Dr. James Barrett Brown.
3. Roundtable Discussion—RHINOPLASTIC SURGICAL PROBLEMS.
   Background for discussion will include the following publications by Dr. James B. Brown:
   1. Plastic Surgery of the Nose..By Dr. James B. Brown and Dr. Frank McDowell.
   2. Review of 800 Articles Covering Plastic Surgery of the Nose..By Dr. James B. Brown, et al., *Journal of Plastic and Reconstructive Surgery*, Vol. 10, Page 149, 1952.

CLINIC WILL BE ARRANGED AT VARIOUS TIMES AND PLACES DURING THE WEEK. THESE WILL BE ANNOUNCED AT THE MEETING.

HEREDITARY HEMORRHAGIC TELANGIECTASIS.*

G. ARNOLD HENRY, M.D.,
Toronto, Canada.

This is a relatively rare disease, characterized by the triad of multiple telangiectases, hemorrhage, and a history of familial occurrence. It has been described under other names, such as multiple hereditary telangiectasis, Osler's disease, Rendu-Osler's disease and Rendu-Osler-Weber's disease.

Recurring epistaxis is usually the first and certainly the most common sympton of the disease. It may be quite severe, and is the complaint which ordinarily prompts the patient to seek medical advice. Frequently the otolaryngologist is the first consultant to see the patient. If he is not familiar with the condition he may be at some loss to separate it from the numerous blood dyscrasias associated with bleeding from the mucous membrane and other parts of the body.

In this paper four new cases are reported, with a study of the two family groups represented. A survey of the literature is presented, together with the author's comments, in an attempt to correlate and clarify existing data on the subject. This is done with the hope that the otolaryngologist may take further interest in this disease and the blood dyscrasias.

HISTORY OF THE DISEASE.

It is not known who first wrote about this disease. In the latter half of the nineteenth century a number of authors — among them Sutton,[1] Babbington,[2] Legg[3] and Chiari[4] — described familial epistaxis associated with angioma of the nasal mucous membrane. Some of Legg's and Chiari's patients, in

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addition to epistaxis, had telangiectases. While it seems likely that some of these early cases were hereditary hemorrhagic telangiectasis, it is difficult to be sure.

Rendu,[5] in 1896, reported the case of a man, aged 52, who had suffered recurrent epistaxis for 40 years. He had telangiectatic areas on the skin of the face and in the mouth, but none were found in the mucous membrane of the nose. The patient's father had had repeated attacks of melena, while his mother and brother had been subject to epistaxis. Many feel that this was the earliest description of hereditary hemorrhagic telangiectasis. Rendu, however, seemed to feel that he was dealing with a type of hemophilia rather than a definite clinical entity.

It was Osler,[6] in 1901, who first established this condition as a clinical entity and separated it from the large and somewhat confusing group of hemorrhagic diseases. In his first report he describes three cases. Two belonged to a family in which epistaxis had occurred in seven members. All three cases had recurring epistaxis and numerous telangiectases on the skin of the face and mucous membrane of the nose, lips, cheeks and tongue.

Weber[8] further classified this condition in several reports. Consequently it is sometimes known as Rendu-Osler-Weber's disease. Brown Kelly[10] and Kofler[11] reported additional cases.

In 1909, Hanes[12] introduced the term hereditary telangiectasis, which has met with more or less general acceptance. Hanes described not only the disease but also the histopathology of the lesions. He commented that the disease is so dissimilar to hemophilia that the presence of the one makes the existence of the other highly improbable.

Fitz-Hugh[13] noted evidence of atavism in four out of 30 families in the literature. Larrabee and Littman[15] took issue with this report, stating that, since the condition is a dominant inheritance, atavism is impossible.

Boston[16] recorded, in 1930, the first gastric hemorrhage due to telangiectasis, reporting three cases in which it occurred.

Nine years later, Renshaw[17] described the gastroscopic appearance of the gastric mucosa. Rumball,[18] Benedict[19] and Goldstein[20] also reported studies of gastric telangiectasis.

Libman and Ottenberg[22] and Fitz-Hugh report hematemesis from this condition. Fitz-Hugh noted that lesions seen bronchoscopically could not be identified four years later at autopsy. Schuster and Hanes stated that individual telangiectases are not necessarily permanent but vary in size from time to time, or may even disappear. Fitz-Hugh[14] further reported splenomegaly and hepatomegaly in some of his severe cases, and noted an intolerance to blood transfusions in some. Snyder and Doan[23] report a case of homozygous hereditary hemorrhagic telangiectasis. They believe this form is lethal. Telangiectasis of the lungs was reported by Brink[24] and Garland,[25] Hanes, Story and Akamatau.[26] Singer and Wolfson[27] described the histopathology of hereditary hemorrhagic telangiectasis. Many other informative articles appeared on this condition, *viz.*, Houser,[28] Figi and Watkins,[29] Voyles and Ritchey,[30] Kushlan,[31] McDonough and Dry.[32]

DIAGNOSTIC AND CLINICAL MANIFESTATION.

This is an hereditary affection, characterized by telangiectatic areas, found chiefly on the skin of the face, neck and hands, and on the mucous membrane of the nose and mouth, which give rise to hemorrhage, either spontaneously or due to trauma. The various blood findings, such as bleeding time, clotting and retraction time, platelet count, white blood count and differential smear, are within normal limits. The blood calcium, icteric index, bromsulfalen liver function and tourniquet test are likewise normal. A hypochromic anemia is usually found, the degree determined by the severity of the bleeding. If the anemia is severe, there will be the usual signs of weakness, palpitation, vertigo, fainting and dyspnea on exertion.

Epistaxis is ordinarily the first and certainly the most frequent complaint of the condition. It usually begins early in the second decade of life. The telangiectases tend to appear in the third or fourth decade, when bleeding often becomes

more troublesome. The gums and hard palate are frequent bleeding sites; less commonly, the fingers. Bleeding may, of course occur from telangiectatic areas anywhere. Gastric, intestinal and rectal bleeding is found quite frequently, and hemoptysis and hemature may occur occasionally. Enlargement of the spleen and liver may happen in some severe late forms of the disease. In telangiectasis of the lung, cyanosis may be present.

CASE REPORTS.

The first three case reports concern a mother and her two daughters. The fourth case was from another family.

Photograph of Mrs. P. C., Case 1.

Fig. 1. Multiple telangiectatic areas of various sizes and shapes are apparent on the face.

Case 1: Mrs. P. C., age 49, was admitted to hospital on Jan. 28, 1947, for investigation of recurrent bleeding from her nose and gums. In addition, her face and other parts of her body were marked by telangiectatic areas.

Family History: This was most remarkable. With the help of relatives, she was able to trace a history of bleeding back through five generations, going back to a great-great-grandmother, who came from Germany. Forty-eight members of family were identified, 31 of which had a history of bleeding. Thirteen of the 31 had telangiectatic spots. Three members of the family died of hemorrhage and anemia. Mrs. P. C.'s three children have recurring epistaxis but have no skin telangiectases. They are still in their twenties.

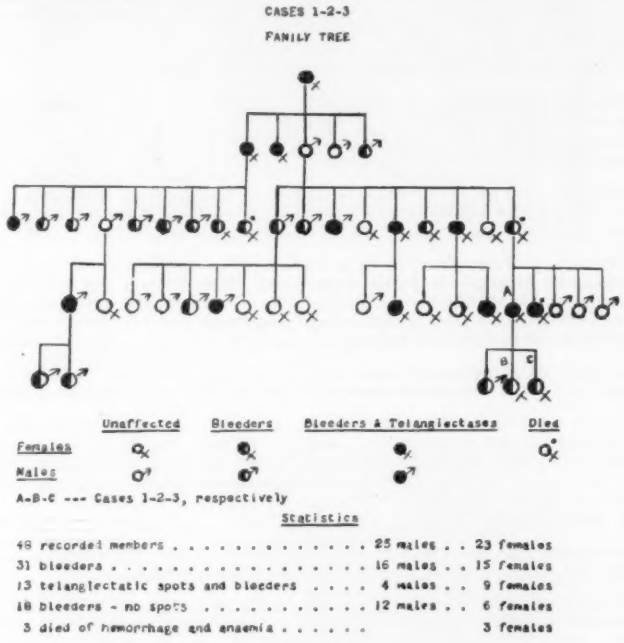

CASES 1-2-3
FAMILY TREE
A
B
C
Unaffected
Bleeders
Bleeders & Telangiectases
Died
Females
Males
A-B-C --- Cases 1-2-3, respectively
Statistics
48 recorded members . . . . . 25 males . . 23 females
31 bleeders . . . . . 16 males . . 15 females
13 telangiectatic spots and bleeders . . . . 4 males . . 9 females
18 bleeders - no spots . . . . . 12 males . . 6 females
3 died of hemorrhage and anaemia . . . . . 3 females

Fig. 2.

Personal History: Epistaxis started as a child and, apart from short intervals, has increased in frequency and intensity. During each of her three pregnancies, bleeding became much less frequent and severe.

Small pinpoint spots appeared at age 35. These spots have increased in number, size and area of distribution since that date.

The menopause began at age 42, and bleeding has been more excessive since.

Prior to a nose bleed, the side of the nose about to bleed often aches severely, or an accompanying occipital headache may be present. After a nose bleed she feels better. Frequently epistaxis is started by a sneeze,

or even by turning in bed too quickly. Any extra nervous strain, or the presence of an unexpected guest, may be sufficient cause. The result is that the patient tends to spend more and more time alone at home.

Physical Examination: The patient is a well nourished female weighing about 140 pounds, and of a cheerful, rather placid disposition. Apart from the telangiectatic areas, the physical examination is negative. She had a previous submucous resection of the nasal septum for nasal obstruction.

The Telangiectases: On the face and neck there are many reddish-purple areas—some raised, some flat—varying in size from pinpoint to one-quarter-inch in diameter. Some have a single connecting vessel, others have several and look like spiders. A few medium-sized areas are present on the palms of the hands, fingers and under the finger nails.

Telangiectatic areas are present on both sides of the nasal septum and are centered chiefly about Little's area. Four spots are present on the hard palate, and on the gum of the upper jaw, near its center, is a large area which bleeds frequently.

There is another area which is about one-quarter-inch in diameter on the floor of the mouth, which, while apparently a telangiectatic area, looks somewhat like a small cyst.

Laboratory Work: The bleeding, clotting and clot retraction time, red blood count, hemoglobin, white blood count, platelet count and differential smear were all within normal limits. The blood pressure, tourniquet test and blood calcium were normal. Her blood type was III. Blood work at later times after bleeding showed secondary anemia of varying degrees.

Biopsy: A telangiectatic area from the thumb and the cyst-like area from the floor of the mouth were removed and sent for histological study.

Treatment: The bleeding area on the gum was injected with 0.5 cm. of 2 per cent phenol in almond oil. Radium, 30 mg., was applied to the left nostril alongside the bleeding areas and left for six and one-half hours. Ten days later the same amount of radium was similarly employed in the right nostril for five hours. Two months later she received another 100 mg. hours in each nostril.

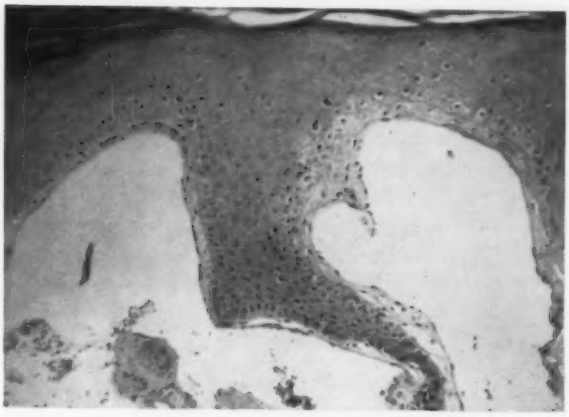
Medically, she was given rutin 50 mg. twice daily with vitamin C. At times, iron and liver injections were given by her physician.

Progress Report: The bleeding area on the gum has not bled since. In the six months' interval after the radium there was very marked improvement. Since then there has been a gradual increase in the amount of bleeding. The patient ceased taking the rutin after about three years. Oddly enough, the bleeding also stopped for about six months and then resumed its regular course.

The telangiectatic areas are more numerous and larger than when first seen, nearly five years ago. One prominent spot on the left eyelid, however, has now disappeared. One or two new areas have appeared in the right nostril over the destruction of the old ones. A new spot has appeared on the thumb near the one removed.

Case 2: Daughter, age 21, August, 1947. This girl has had mild attacks of epistaxis for 10 years.

Physical Examination: This is negative except for three telangiectatic areas on the left side of the nasal septum in the region of Little's area.



Microscopic Study—Biopsy Specimen, Case 1.
Telangiectatic Area from Thumb.

Fig. 3 × 100. Section shows two large, thin-walled blood vessles, lying under a thick layer of squamous epithelium. Overlying the epithelium is an extremely thick covering of keratin. Clusters of red blood cells can be seen within the blood spaces.

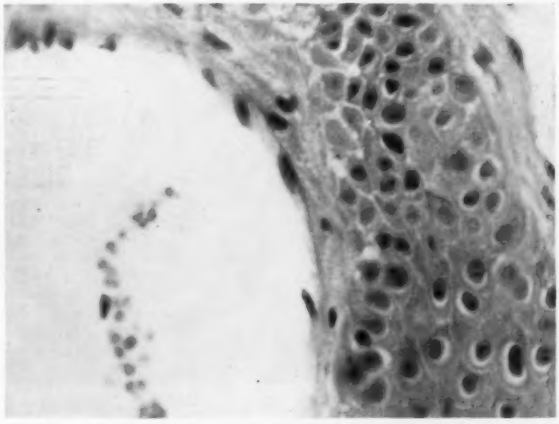
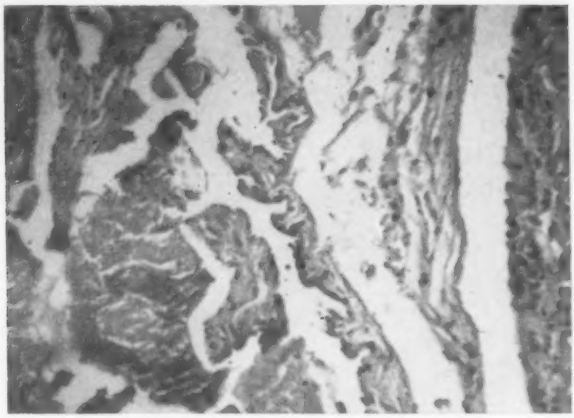


Fig. 4 × 500. A portion of one of the blood vessels is shown under greater magnification. The blood vessel wall consists only of a dense, fibrous tissue coating.



Microscopic Study—Biopsy Specimen, Case 1.
Telangiectatic Area from Floor of Mouth.

Fig. 5 × 100. Section shows numerous, irregularly dilated, thin-walled blood vessels.

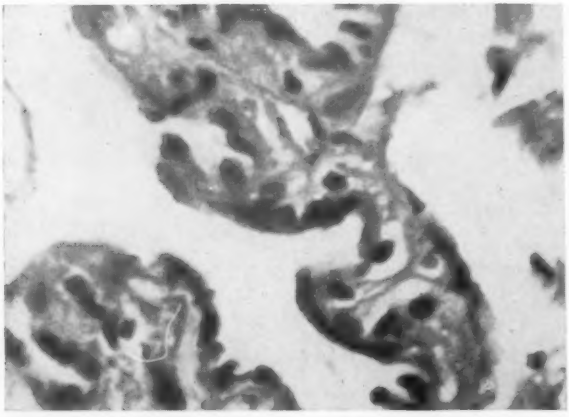


Fig. 6 × 500. Higher magnification shows an irregular, dilated blood vessel lined, for the most part, by a single layer of endothelium. The wall consists of dense, fibrous connective tissue which blends with the supporting stroma. There is no muscular or elastic tissue visible in the wall of this blood vessel.

Treatment: Electrocautery was applied to area on two occasions. Her physician gives her occasional iron therapy.

Progress Report: Occasional nose bleeds, which do not amount to much —would say condition improved over the last five years.

Case 3: Daughter, age 20, August, 1947. This girl has had occasional nose bleeds for three years.

Physical Examination: This was negative, except for two small angiomatous areas on the left side of the septum near Little's area.

Treatment: Electrocautery on two occasions—iron therapy.

Progress Report: There has been some improvement in the amount of bleeding over the last five years.

Case 4: Mr. W. J., age 62, was referred to my office in August, 1948. He had had recurring epistaxis since the age of 14. These attacks were not too severe until spots started to appear on his face around 30 years of age. The hemorrhages increased with age and he had, on at least four occasions, been so thoroughly bled out as to require blood transfusions.

Family History: One brother, has telangiectases and recurring epistaxis. Unfortunately, he did not know much about his family, other than that he believed his father and mother were free of the condition.

Physical Examination: His physician stated that, outside of the telangiectasis and poor peripheral circulation, his physical examination was essentially negative. Bleeding, clotting time and differential smear were essentially negative. His blood group was type I. He had a mild secondary anemia.

Telangiectases: This man's face and neck were heavily marked with simple, cherry and spider forms of telangiectases. A few appeared on the palms of his hands, on the fingers and under the finger nails. One spot was present on his left eardrum. There were four or five grouped areas on each side of the nasal septum about Little's area. There were two spots on the hard palate.

Treatment: Fifty mg. of radium were inserted over the bleeding area on the left side of the nose for five hours. A similar dose was administered to the right side two weeks later.

This patient was not seen again, since he came from some distance, but his physician tells me there was a very marked improvement, in that he had fewer and less severe attacks of epistaxis. This improvement was maintained until his death about a year ago from gangrene of the foot. He had refused amputation.

INCIDENCE.

It is difficult to estimate the incidence of this disease. Kushlan,[31] in 1946, stated that there were over 175 families recorded, with more than 1,000 members. In 1950, Garland listed the number of families at 244, with 648 affected males and 703 affected females. There were 64 of unstated sex.

Some writers have been inclined to add a few borderline cases to the group. It seems likely that many cases have gone unrecognized and that many more, while recognized, have not been reported. The author agrees that it is a relatively rare disease but believes it occurs more commonly than reports in the literature indicate.

HEREDITY.

Evidence of this condition in many family trees is indisputable proof of its hereditary nature. It is not sex-bound, but is capable of transmission by either side of the family to either sex. There have been instances where it has skipped a generation or two only to reappear in a later one. It has been pointed out that, since this condition can be transmitted by either sex, it is a dominant inheritance, which does not admit of atavism. It may be that, where atavism seems apparent, the individuals have the disease in a mild or abortive form.

Since most reported family groups concern only three or four generations, there is some feeling that this may be evidence of self-limitation of hereditary transmission. On this continent, at least, it would be unusual to trace a family beyond three or four generations. Teahan,[24] however, has reported the condition through six generations, and Case 1 in this paper presents five generations of bleeders.

Snyder and Doan[23] report an unusual case worthy of study. A full-term, eight-pound female infant was born to young parents, both of whom had hereditary hemorrhagic telangiectasis, as did two grandparents—one on either side. The infant was normal at birth, except for an area about two inches in diameter over the left chest in which the superficial vascular bed was dilated. On the fifth day, telangiectatic spots began to appear. First signs of hemorrhage appeared at 28 days in the urine and stools. The infant died, aged two months and 18 days.

At postmortem, evidence of telangiectasis or bleeding was present in the skin, mucous membrane, and the majority of

organs and tissues throughout the body. The bleeding interfered with the gastrointestinal and urinary functions. The fatal outcome was due chiefly to the profound anemia.

The authors feel that this disease occurred in the heterozygous form in both parents. In the infant, the opportunity occurred for it to be transmitted by both parents—homozygous form. They believe the homozygous form is lethal.

Speculating as to the question of occurrence in other children, they reason thus: if we let T represent the semidominant lethal gene for telangiectasis, and t its recessive form, then three gene types are likely—TT, Tt and tt. Since the parents are Tt, we would expect the offspring to show TT, two Tt, and tt. The lethal gene, therefore, would occur once in four. There would be two children with classical telangiectasis and one unaffected

Singer and Wolfson[27] give some further evidence of the possible penalties of intermarriage in such families. A girl, third generation with telangiectasis, married her first cousin, third generation, who did not have telangiectasis. There were two children — the first a stillbirth, the second exhibited a complete spina bifida. There were other reports of anomalies of the vertebral column.

Fitz-Hugh[14] noted that, in those of his patients who were blood-typed, the group was IV. He wondered if this was a characteristic of the disease. Goldstein had one case, a type III. Two of the cases reported by the author were types III and I, respectively. Reports from a confrere[40] showed his cases to be other than type IV. Apparently, the blood grouping of individuals with this disease is not limited to any one type.

THE TELANGIECTASES.

The coexistence of spider angiomata, cirrhosis of the liver, and other chronic liver disorders has been known for many years. This may be associated with dilated venules on the neck and face. Indeed, many otherwise normal individuals may have areas of telangiectases, in appearance not much different from the disease, on various parts of the body.

In hereditary hemorrhagic telangiectasis, the lesions are found most frequently in the mucous membrane of nose, gums and hard palate, and on the skin of the face, ears, palms of the hand and the fingers, particularly under the finger nails. Any of the other organs and viscera may be involved. The lesions tend to be most numerous in areas of constant irritation or trauma.

Typically, the telangiectatic area is purplish-red, pinpoint to one-quarter-inch in size, sharply demarcated, elevated or flat. If a single arterial vessel connects with it, it is called a cherry angioma: when several vessels are noticeable, it looks like and is called a spider angioma. There are three typical forms: a node or spot, cherry-like forms and spider forms. They will blanch on pressure, and the color returns when the pressure is removed. The color may be due to peripheral factors, such as slowing of the circulation in dilated vessels with consequent increased loss of oxygen. It may be due to increased visibility of the capillary and venous network. More rarely, it may be due to pulmonary abnormalities, such as arteriovenous anastomosis or fistula, as described by Brink.[24]

There is some controversy as to whether the telangiectatic areas are capable of disappearance. Schuster and Hanes, and Fitz-Hugh feel that this occurs. Bean feels that the lesions do not disappear and can always be found, except when they fade with the profound anemia which follows hemorrhage. In our experience, when a lesion is removed for biopsy, it is almost impossible to see it with the naked eye. Pathologists have great difficulty finding the lesions at postmortem unless the areas were ringed during life. It is evident that the color of the lesions is not due to clots or stagnant blood. In Case 1 in this paper one of the lesions — a single, prominent spot on an eyelid — seen in 1947, could not be found five years later.

HISTOPATHOLOGY OF THE LESIONS.

The histopathology of the telangiectatic lesions was well exemplified in the two sections taken from Case 1. They are composed of dilated small vessels which, histologically, are

made up of a single layer of endothelium with a conspicuous deficiency of the muscle and elastic layers of the vessel walls. Because of this deficiency, they have a tendency to bulge toward the surface. This is the only difference between a lesion in hereditary hemorrhagic telangiectasis and a simple red angioma which has never bled.

THE HEMORRHAGIC TENDENCY.

In the hereditary hemorrhagic diathesis group, the cause of hemorrhage falls roughly into three main categories:

1. Disturbance in the plasma elements.
2. Alteration of platelets — quantitative or qualitative.
3. Disorder of capillary structure and function.

With the exception of secondary anemia, the blood in hereditary hemorrhagic telangiectasis is normal to all tests, thus ruling out groups I and II. The one known cause of bleeding is the weakness of the thin-walled blood vessels in the telangiectatic areas. We do not know the mechanism which brings about this hereditary manifestation. Individuals with this disease, apart from the telangiectatic areas, show no greater tendency to bleed than other individuals.

MORTALITY.

Some authors feel that this disease does not ordinarily inhibit development nor shorten the span of life. One can find cases in the literature and in the author's family group which seem to bear out this contention. On the other hand, there seems to be a proportionately large number in the author's group who died in the 40's of cerebrovascular or other troubles. It would be reasonable to expect that many not dying of the disease would be cut down before their time as a result of lower resistance due to frequent loss of blood. Actual mortality to the disease is difficult to determine. Death, when it occurs, is usually from the profound anemia of one or repeated hemorrhages. In the group presented by the author, three died out of 31 cases—9.7 per cent. Three out of 11 of Houser's group of cases died — 27 per cent.

These figures are somewhat higher than others in the literature, and the 4 per cent suggested by Houser may be more accurate.

DIFFERENTIAL DIAGNOSIS.

Hemophilia, pseudohemophilia, purpura hemorrhagica, acute leucemia and aplastic anemia should be included in the group.

Hemophilia occurs in males only and is transmitted only by females. Constant liability to excessive hemorrhage from slight injury and hemarthrosis are common symptoms. Blood work shows delayed coagulation time, but bleeding time, platelet count and clot retraction are normal.

Pseudohemophilia — epistaxis, spontaneous ecchymosis. Prolonged bleeding after operation and spontaneous bleeding from mucous membrane are common symptoms. Blood work shows increased bleeding time. Tourniquet test may be positive or negative. Clot retraction may be defective in some cases.

Purpura hemorrhagica occurs in either sex. There is usually a history of cyclic episodes of hemorrhage from mucous membranes, associated with ecchymosis over the body and extremities. Blood work — platelets are reduced and bleeding time is prolonged. Clot retraction is delayed or absent, but coagulation time is usually normal.

Acute leucemia occurs in both males and females. There is a rapid onset of anemia with fever and ulceration of the mucous membranes. Frequently there is hemorrhage from gums, nose, stomach and rectum. The spleen is enlarged. The diagnosis is usually made from the white blood count and smear, where the distorted cell picture and immature cells are found.

Aplastic anemia — history of weakness and anemia prior to hemorrhage. The spleen is not palpable. Purpura and hemorrhage may occur anywhere. Blood picture — very low red and white cell count. The platelets are reduced and bleeding time is prolonged. Clot retraction is delayed.

Bleeding from esophageal varices associated with cirrhosis of the liver might be a source of confusion.

The differential diagnosis of hereditary hemorrhagic telangiectasis is not too difficult if one remembers that the blood is normal, except for secondary anemia. There is the diagnostic triad of symptoms — bleeding, usually nasal, multiple telangiectases, and hereditary characteristic.

TREATMENT.

The histopathology of the telangiectatic lesions and the lack of abnormality in the blood, except for secondary anemia, suggests treatment in two directions. The first will concern itself with the hemostasis and destruction of the bleeding lesions where possible. The second will include treatment for any secondary anemia, the patient's health generally, and therapy which might improve the strength of the telangiectatic vessel walls.

Pressure directed against the bleeding areas is the best immediate means of hemostasis. In the nose, where the majority of these hemorrhages occur, nasal and postnasal packing will usually suffice. In some cases, the patient would achieve considerable peace of mind by carrying a rubber bag inflation plug, as recommended by Hurst and Plummer.[35] This may consist of a finger stall with a soft rubber catheter thrust to its end and tied at the opening around the catheter. The patient can carry it lubricated and, when required, he can install, inflate and clamp it himself. Later treatment must be directed where possible toward the destruction of the offending telangiectatic lesions. This may be done in a number of ways — electrocautery, chemical cautery by surface application or injection, electrolysis or radium. Radium, electrocautery and injection of phenol were used in our cases.

Continuous rutin therapy[31] seems logical. Iron and vitamins B and C may be used continuously or from time to time. Liquid petrolatum or some oily nose drop should be used once or twice daily to avoid crusting or keep it at a minimum. Hesperidin (vitamin P[40]) and snake venom[38] have been ad-

vised to improve the integrity of the blood vessel endothelium. It is important to keep the secondary anemia at a minimum. Where the blood loss has been great, transfusion should be given. The patient should have the constant care of his family physician, who will watch the hemoglobin and red blood count.

Paul,[36] McKay and McKenty,[37] and Houser have used radium to destroy the lesions with apparently satisfactory results. Figi and Watkins[38] feel that the after-dryness, crusting and bleeding, following the use of radium, makes it unsatisfactory. The author suggests that the method of destruction should be governed by the site and number of lesions. Single lesions on the gums, hard palate and fingers may be injected satisfactorily with a sclerosing solution. Where multiple bleeding areas are close together on the nasal septum, radium seems to be a satisfactory method of treatment. Less numerous and severe lesions probably should be treated first with electrocautery. Lest one be dogmatic, it is apparent that, in some cases, there appears to be no lasting method of stopping the bleeding. This is due to the return of the old lesion or the appearance of a new one.

COMMENT.

The study of a disease such as hereditary hemorrhagic telangiectasis leaves one very humble. We are aware of its essential characteristics, but do not know why many of them occur.

We would like to know the mechanism which causes the loss or thinning-out of the muscle and elastic tissue in the telangiectatic vessel walls. Why does bleeding seldom occur before puberty, and why do spots not appear until the mid 30's? What happens when a spot disappears spontaneously? Case histories often show cessation of bleeding for several months or longer. In addition, the author's Case 1 reported considerably less bleeding during each of her three pregnancies. So far, we do not know the reason for these occurrences. We do know that trauma is an important factor in bleeding,

and we suspect it may have some influence on the cause and site of the telangiectatic areas. An endocrine factor in this condition would also seem to be a reasonable supposition.

While this disease is relatively rare, one notes with surprise the 31 cases in one family tree, reported by the author. Since the mortality is not high, there must be some form of self-limitation in the inheritance of the disease. On the subject of inheritance, the danger to offspring seems obvious in the intermarriage of individuals in affected family groups.

Hereditary hemorrhagic telangiectasis in an individual does not preclude the presence of other conditions. There is a tendency, in bizarre diseases such as this, to include any unusual manifestation as an essential part thereof. Perhaps some of the liver, spleen and lung manifestations may turn out to be associated conditions. Careful reporting of additional cases in the literature should do much to increase our knowledge of this interesting disease.

SUMMARY.

1. Hereditary hemorrhagic telangiectasis became a clinical entity when Osler described the condition in 1901.

2. The disease is transmitted by, and occurs in, either sex.

3. The diagnosis depends upon the triad of bleeding, multiple telangiectases and hereditary characteristics.

4. Epistaxis is the most common symptom, but bleeding may occur wherever a telangiectatic area is present.

5. Four cases are reported by the author. Three of these occur in a family with five generations of bleeders.

6. Since the blood is essentially normal in this condition, bleeding occurs because of a structurally weak blood vessel wall in the telangiectatic area.

7. Estimates of some 1,500 people with this condition to 1950 are probably very low. Mortality figures run 4 per cent, or more, of bleeders.

8. Hereditary hemorrhagic telangiectasis can be identified from the blood dyscrasias or other diseases by the normal blood and the triad of symptoms.

9. Treatment, where possible, is to stop the bleeding and destroy the offending telangiectatic area. Additional therapy should be given to combat the secondary anemia, increase the integrity of the blood vessel walls and, generally, improve the patient's health.

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CRITICAL EVALUATION OF NOISE AUDIOMETRY.*

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INTRODUCTION.

The phenomenon of masking is qualitatively evident in everyday life whenever we encounter difficulty in hearing some sounds because of the presence of other sounds. Masking has also been observed quantitatively in laboratory experiments, primarily for two reasons: *1.* the data are useful as basic information about the auditory system; *2.* on the basis of these experimental results, we can now predict fairly well the degree of difficulty that persons with normal hearing will have in hearing speech and tones in the presence of various noises.

Of more recent origin is an interest in masking as a test for certain kinds of diagnostic information in clinical otology. As far as this clinical interest is concerned, it appears that Langenbeck[10] was the first to be concerned with possible differences among measures of masking for persons with different kinds of hearing loss. With a presumedly similar but unstated aim, Weersma[18] made further measurements of this kind, followed soon thereafter by Huizing.[7] These three workers were concerned with the audibility of pure tones in the presence of various noises. Their experiments, to be reviewed presently, were somewhat equivocal because of apparently large differences in the spectral composition of their noises. More recently Langenbeck[12-15] has employed a thermal (white) noise with a uniform spectrum so that his clinical

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results can be compared with laboratory results on normal listeners. Huizing,[8] Kietz,[9] and Zangemeister[20] have also added recent information on masking by white noise.

Although some of the details are still missing, the quantitative specification of masking for normal listeners has been fairly well worked out. Experiments on the audibility of pure tones in the presence of other pure tones were reported in 1924 by Wegel and Lane[19] and have not been substantially altered by further research except that some of the difficulties of measurement that arose when the frequencies of the masked and masking tones were similar have been avoided in part by Egan and Hake.[1] Apparently the first investigation of the audibility of pure tones in the presence of noise was made by Galt[4] in 1929, but he used "natural" noises with rather bizarre spectra. In 1937 the classic experiments of Fletcher and Munson[3] gave us not only information on the masking of tones by a noise whose spectrum was carefully tailored to provide equal masking at all frequencies, but also provided data on the basis of which Fletcher[2] could recognize and calculate the widths of critical bands. These measurements of masking by thermal noise were corroborated and extended later by Hawkins and Stevens.[5]

In a recent revision of his 1929 book, Fletcher[2] has summarized his calculations on the relation between loudness and masking. In view of the intimate relation between masking and loudness in normal hearing, it is not surprising that masking should be thought of as an important phenomenon to be investigated in the clinic, especially since the abnormal loudness functions that are reported under the rubric of recruitment have became such important diagnostic tools.

In this paper we present certain basic conclusions of the experimental literature with regard to masking in normal listeners and their relations to analogous phenomena in various types of hearing loss; furthermore, we wish to analyze the relations that have been claimed by certain of the authors mentioned above who have employed masking in clinical audiometry. Finally, we report results on the masking of

pure tones by white noise which show that the various diagnostic categories commonly used in otology cannot possibly be distinguished on the basis of masking data.

NORMAL MASKING BY WHITE NOISE.

The fact that a person with normal hearing may appear to have a hearing loss at certain frequencies on the audiometer when his hearing is tested in a noisy room shows how the masking effect of a noise can be measured. It is clear that different noises will have different effects: a noise with most of its energy in the low frequencies will impair the hearing for low frequencies more than it will for high, and vice versa. Because of a certain simplicity that is introduced into the results, our interest can be focused on the influence of a particular kind of noise, called "white noise," on the audibility of pure tones. What makes this noise white, an analogy to white light, is the fact that all frequencies have the same amplitude. If we were to make a harmonic analysis of a white noise, we would find that the energy present at 100 cps would be exactly the same as the energy at 1000 and 5000 cps or at any other frequency. The very well shown by Hawkins and Stevens,[5] whose results effect of such a noise on the audibility of pure tones was are given in Fig. 1.

First these authors found the absolute threshold for pure tones at various frequencies in the quiet. These are shown as the lowest curve in Fig. 1, plotted on an absolute scale (rather than the "hearing loss" scale of the audiometer). These experimenters then measured again the thresholds for these same pure tones, this time in the presence of the white noise at a particular intensity level. The experiment was repeated at each of eight noise levels, and the results of each of these experiments are given as one of the eight curves above the quiet curve in Fig. 1. Actually the procedure is no different from measuring a person's pure-tone threshold in the quiet, except that the thresholds are now measured with a noise that is mixed electrically with the tone.

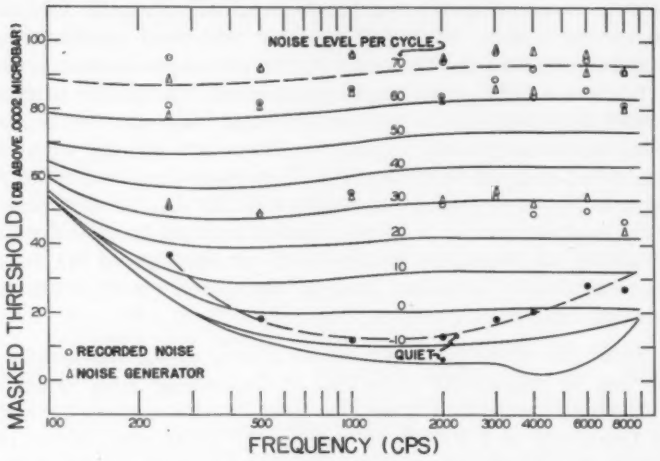


Fig. 1. Pure-tone thresholds in the presence of noise as a function of frequency. The solid curves represent the data of Hawkins and Stevens.[5] The dashed curve connecting the solid symbols shows quiet thresholds measured in the present experiment. The open symbols show masked thresholds measured in this experiment at noise levels approximating those indicated on the nearest curves. The top dashed curve represents an extrapolation of 10 db from the curves of Hawkins and Stevens.

The masking effect of the noise at very low levels is evident only for middle frequencies, where the ear is most sensitive. As the noise level is increased above 30 db per cycle, however, the equal-masking contours are parallel, showing that at all frequencies a 10 db increase in noise level corresponds to a 10 db increase in the masked threshold. On the basis of this fact we can state that the relation between the masking of a pure tone by a noise and the intensity of the noise is linear, above the low levels.

MEASUREMENT OF NOISE.

In order to relate these experimental data to the clinical reports and our own results that follow, we must discuss the way in which the noise level is measured. In order to specify the sound pressure level that is introduced into the ear by an earphone, we ordinarily rely upon a frequency-response characteristic which tells us how much sound pressure an earphone will produce in an artificial ear at each of

several frequencies when a fixed electrical voltage is applied to the earphone. The earphone that was used by Hawkins and Stevens, like the one used in our own experiments, gives a fairly constant sound pressure level of 105-106 db (above 0.0002 microbar) up to 6000 or 7000 cps when 0.1 volt is applied to the earphone. Thus, we know that a pure tone at any frequency up to 7000 cps will produce a sound pressure level in the ear canal of approximately 105 db if at the same time we measure the voltage across the phone to be 0.1 volt. Similarly, if the noise voltage reads an average of 0.1 volt, we can specify the noise level as an overall sound pressure level of 105 db.

Since the white noise has equal energy at all frequencies, we can be reasonably sure that if the overall level from 0 to 7000 cps is 105 db, then the level in each one-cycle band of frequency is 1/7000th of this level. Since the 105 db is a logarithmic quantity, we do not divide it directly by 7000 but rather subtract from it 10 $\log_{10}$ 7000, or 39 db. Thus, in Fig. 1, where the top solid curve refers to a noise level per cycle of 60 db, we can infer that the overall noise level was 99 db. These two measures, *overall level* and *level per cycle,* specify the intensity of the noise on an absolute scale.

There are other ways of stating the intensity of a noise, which do not require the use of complicated measuring instruments. One of these is "sensation level," that is, the level of noise in db above the threshold for the noise. For example, the overall level of a white noise that can just barely be heard is about 10 db (above 0.0002 microbar) . This allows us to specify a noise whose overall level is 100 db as a noise whose sensation level is 90 db. Although it is not clearly stated, we believe that sensation level is the measure of noise intensity used by both Kietz[9] and Zangemeister.[20]

There is one other measure of noise that can be used, and this is the one employed by Langenbeck.[11-16] Suppose we present a tone to a listener at a sound pressure level of 50 db. Leaving the tone's intensity fixed, we now vary the intensity of a white noise until it just barely masks the tone,

i.e., the tone is just barely audible in the presence of this particular noise level. We do not know what the noise level is, but we know that it just masks a 50-db tone. This unmeasured level is what Langenbeck calls a 50-db noise. It is almost exactly the "effective level" of the noise which is the sound pressure level of the critical band of the noise that applies to the pure tone being masked. Briefly, a critical band of noise is a band of frequencies immediately surrounding the pure tone that is being masked, whose width is such that the overall energy in this band is equal to the energy of the tone that is being masked (see Hirsh,[6]). It is clear from the data of Fletcher and Munson[3] and Hawkins and Stevens[5] that the critical band is not constant for all frequencies but rather is at a minimum around 400 cps, rising slightly toward the low frequencies and much more toward the higher frequencies. The critical band is about 20 db wide at 100 and 2000 cps, dropping to a minimum of 17 db at 400 and rising to 27 db at 7000 cps. Again this width of a band of frequencies is given in db instead of cps (W (db) = 10 log. W (cps)).

We may tie these four measures of noise together very simply for white noise by looking at Fig. 1, top solid curve. The noise level per cycle is given as 60 db. In the 0-7000 cps band this corresponds to an overall noise level of about 99 db. This same noise level can be referred to as a sensation level of 89 db, since the threshold for this noise is 10 db sound pressure level; furthermore the approximate effective level for this noise is 80 db, the approximate threshold of the pure tones at all frequencies in the presence of this noise.

In our own experiments we employed a noise in the band 20-7000 cps whose overall level was 100 db (above 0.0002 microbar). The results of our measurements with normals, to be described presently, are shown by the symbols near the corresponding curves of Fig. 1. It may be noted that our normal thresholds lay 1-3 db above those of Hawkins and Stevens, partly because, for example, our 100 db overall level corresponds to a level per cycle of 61.5 db instead of the 60 db of the top solid curve.

We shall describe some of Langenbeck's procedures in the next section, but it may be pointed out here that the noise employed in his later papers[12-15] was not exactly a white noise but rather it was noise that had slightly more energy between 100 and 2000 cps and slightly less above 2000 cps in order that his masked thresholds would all fall on a straight line instead of curving slightly upward as do the contours in Fig. 1. We may say that his noise, instead of having equal energy per cycle through the frequency range, had equal energy per critical band throughout the frequency range.

REVIEW OF NOISE AUDIOMETRY.

Different kinds of noise, used as sources for masking, have been employed in the clinic for some time. Otologists and audiologists are familiar with the masking noise or buzz supplied in most audiometers to be used for blocking one ear while testing the other. This is not, however, the use under discussion here although the present results do have some implications for masking used in this way. We are interested in the threshold for pure tones measured in the presence of noise when both tone and noise are presented to the same ear. Clinical interest in this phenomenon probably can be dated back to 1680 when Willis reported that persons with hearing loss appeared to hear better in noise than normals. It was not until the latter part of the 19th century that it became clear that the noise influenced not so much the hearing of the listener as it did the speaking level of the examiner. Then three years after Galt[4] reported the influence of various noises on normal pure-tone thresholds, Langenbeck[10] studied the effect of noises on the audiograms of four normal persons, 10 with perceptive hearing loss and 11 with conductive or mixed hearing loss. His most frequently used noise at that time came from an electric fan operating at 1400 rpm. Both noise and tone were produced in the field around the listener's head. Langenbeck noticed first that the masked thresholds were somewhat higher for the hard-of-hearing cases than for his four normals, but it appears that the greatest deviations between these threshold curves

occurred where the hearing losses in the quiet were even greater than those produced by the noise on the normal listeners. He also pointed out that the noise had a more severe masking effect on the perceptive than on the conductive cases although the difference was very small.

Weersma[13] employed the noise of an electric buzzer and thermal noise with and without filters to mask pure tones, both tones and noise being presented in the field. He concluded that in nerve cases the masking effect was very great at high frequencies, but in the case reported in the article this effect again may have been due only to the large hearing loss that this case had to begin with in the quiet. The noise did appear to affect the hard-of-hearing listeners more (higher thresholds) than normals.

In 1942 Huizing[7] proposed that thermal noise might be used to detect recruitment in cases where there were hearing losses in both ears. His results showed more masking at low noise levels for perceptive cases than for normals and less than normal masking above 50 db sensation level of noise for conductive cases. The relation between masking and recruitment was stated quite explicitly by Huizing: the masking increases more rapidly with noise level for cases with recruitment than for cases without.

Langenbeck[11] returned to noise audiometry with an electric noise generator. In this paper the spectrum of the noise was still irregular, and the masking did not increase linearly with the intensity of noise. Even so, he claimed to be able to divide the group of perceptive hearing losses into two categories of masking behavior, one associated with more masking than normal, the other yielding roughly the same masking as normal.

The four papers referred to above will not be discussed in detail with regard to noise measurement or procedure because the spectra of the noises were quite different. Langenbeck[12] finally succeeded in generating a noise that would produce the same threshold for pure tones at different frequencies. He then labelled the noise intensity as the intensity of the pure

tone (at any frequency) which was just masked by this noise in the normal listener. In this and the following three papers[13,14,15] Langenbeck's conclusion relative to differential diagnosis was that conductive and cochlear deafnesses show normal masking behavior whereas nerve-fiber deafness is characterized by masking thresholds that are 10-15 db higher than the normal threshold in the same noise.

In addition to this general conclusion, Langenbeck provided additional clues about differentiating certain types of hearing loss. For example, he stated that the threshold for noise (intensity measured as stated above) always corresponds to the best (lowest) pure tone threshold in terms of absolute sound pressure level for all conductive cases and those perceptive cases showing less than 30-40 db hearing loss. In more severe cases of perceptive deafness the threshold for noise may be better than that for the best pure tone, sometimes as much as 15-20 db.[15] This observation was reported to be consistent through 263 cases of study and it was concluded that in mixed deafness this difference in threshold between noise and best pure tone reveals the amount of perceptive component.

In two of Langenbeck's papers[14,15] the masked thresholds for cochlear deafness showed behavior similar to his nerve-fiber cases. His explanation was that the fatiguing effect of the noise is substantial in cases of cochlear deafness, and the measured masked threshold should be referred, not to the threshold in the quiet, but rather to the quiet threshold that is measured immediately after the noise is turned off.

Most of the above findings were either repeated or confirmed in Langenbeck's recent book.[16] In this latest source, however, he did not report any observations regarding a difference between noise and best-pure-tone thresholds that is to be considered indicative of the perceptive component in a mixed deafness; rather, he interpreted any such threshold difference as being within the error of measurement of the pure-tone threshold. This difference, therefore, might be useful in the detection of malingering but not in differential diagnosis.

Kietz[9] and Zangemeister[20] employed a novel technique for measuring similar masking behavior. They selected a signal tone (2000 cps) whose masking by white noise was to be studied. The tone was presented first in the quiet and then the noise was gradually and automatically increased in a Békésy-type audiometer in which the patient recorded his own threshold on a paper chart, the abcissa of which was not frequency but rather the sensation level of noise. A normal listener generated a straight line with a slope of one. Kietz[9] showed one example of a case of perceptive deafness with recruitment in which the masked threshold increased less rapidly than did the noise level, indicating that this patient could hear pure tones in the presence of noise relatively better than normal.

Zangemeister[20] showed several results observed with the same technique. He gave four figures illustrating the behavior of four conductive losses: one behaved like the normal, one showed less masking than normal, and two showed more masking than normal. Two of the graphs illustrated the behavior of recruiting ears with "labyrinth hypertension;" the masked thresholds in these cases increased more rapidly than noise intensity, indicating that the presence of recruitment affords worse hearing in the presence of noise than normal. These two recruitment cases agree in principle with the hypothesis stated earlier by Huizing,[7] but show results exactly opposite to those of Kietz.[9] Finally, Zangemeister[20] showed two more cases, one with "isolated cochlear damage" and the other with nerve-fiber degeneration. The author claims to be able to make this distinction on the basis of audiometric results, but the masking functions for the cases presented appear to be exactly the same.

In 1952 Huizing[8] wrote again on noise audiometry, this time presumably with his white noise under better control because, in contrast to his 1942 paper,[7] the relation between the normal masked threshold and the noise level was now linear. The conductive cases behaved like the normal but the recruiting ear showed 10-15 db more masking throughout all the intensities. We must point out again that

this conclusion, which agrees with Zangemeister, is exactly the opposite of the conclusion reached by Langenbeck and Kietz.

We have done an injustice to all of the above quoted authors by not reviewing their works in more detail. Also, it should be pointed out that we have restricted our discussion to the masking of tones by noise. We have not, therefore, included reference to some interesting clinical papers on the masking of tones by tones and the masking of speech. We have not, however, misrepresented the implications of their findings, nor their general conclusions. When we take this literature on noise audiometry as a whole we must conclude that the results are somewhat chaotic. One can associate almost any pathological masking behavior with almost any kind of diagnostic category.

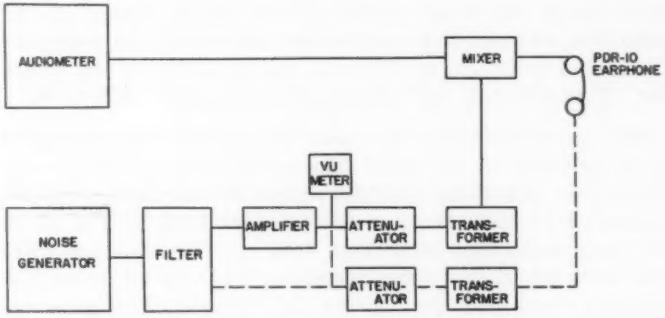
There is one theoretical contribution that should be pointed out and it has to do with the concept of the critical band. Fletcher[3] proposed that the only frequencies in a wide band of noise that contribute significantly to the masking of a pure tone are those frequencies adjacent to the frequency of the tone. The width of this adjacency is the critical band width. Such claims as have been made by the above quoted authors imply that a pathological ear can do different things with the noise and with a tone, so that the masking effect of the former upon the latter may be different in different types of pathology. Even if we make the assumption of a unique relation between masking and loudness we must conclude, on the basis of the critical-band concept and in opposition to Huizing, that the loudness of a tone increases at the same rate as the critical band of noise that just surrounds the tone. When Huizing[7] states for example that the masking increases more rapidly than the noise level in cases of recruitment, this suggests that the loudness of the noise increases more rapidly than the loudness of the tone.

By now the concept of the critical band is not mere theory but rather has been substantiated by at least two series of masking experiments (Fletcher and Munson,[3] Hawkins and

Stevens[5]) and its generality has been attested by the close relations among *1.* the width of the critical band, *2.* the contribution of different frequency bands to the intelligibility of speech, *3.* the mel scale of pitch, and *4.* the size of the just-noticeable change in frequency as a function of frequency.[17] On theoretical grounds alone, then, we would predict that so long as the masking data are obtained at levels well above the absolute threshold in the quiet, the data must all be alike no matter how much or what type of hearing loss exists. This hypothesis has been tested by the experiments to be reported.

EXPERIMENTAL PROCEDURE.

Figure 2 shows a block diagram of the apparatus used to measure thresholds in the quiet and in the presence of noise. The source for pure tones was a standard screening audiometer (ADC-Microtone Type 53-C). This audiometer was



BLOCK DIAGRAM OF THE APPARATUS

Fig. 2. Block diagram of apparatus used to measure pure-tone thresholds in the quiet and in the presence of different levels of noise. (The dashed line indicates the circuit used for contralateral masking.)

fitted with Permoflux (PDR-10) earphones and was recalibrated* in terms of the sound pressure levels that correspond to 0-db hearing loss on the audiometer which are given

*We are indebted to Dr. R. W. Benson, who made these measurements at Central Institute for the Deaf.

in Table 1. In order to mix the noise electrically with the tone so that both would be presented to the same ear, an electrical mixing network was provided. When this network was inserted the sound pressure levels corresponding to 0-db hearing loss were smaller; these values are also given in Table 1. The procedure for measuring the pure-tone threshold

TABLE 1.
SOUND PRESSURE LEVELS OF ADC AUDIOMETER AT 0-DB HEARING LOSS.
(Db Above 0.0002 Microbar)

Frequency	250	500	1000	2000	3000	4000	6000	8000
Normal Use.....	40	23	14	17	21	23	22	28
With Mixing Network	34	18	11	12	16	18	18	24.

in the quiet was a standard clinical one in which the tone was interrupted manually and the threshold was approached both from above and from below at least three times. If the difference between ears was greater than 40 db, the worse ear was tested while 100 db of noise was presented to the better ear. This obtained for both quiet and masked thresholds.

The same procedure was used to measure the threshold in the presence of the noise. The overall level of 100 db was obtained by providing a voltage across the earphone that corresponded to an average sound pressure level of 100 db in the frequency range from 20 to 7000 cps. An electrical filter was provided in the noise generator that cut off noise frequencies above 7000 cps. During the course of these measurements two noise sources were used: one was a magnetic-tape recording of a white noise and the second, used in most of the measurements, was a noise generator with the filter inserted. Measurements of the threshold for pure tones on normals provided evidence that the masking effects of these two different sources were the same, except for small differences at 250, 3000, and 6000 cps (see Fig. 1).

The results for eight normal ears tested in the quiet and in the presence of noise at overall sound pressure levels of

70, 100, and 110 db are given in Table 2 and are shown by the symbols in Fig. 1. It should be borne in mind that the sound pressure levels per cycle of noise indicated on the

TABLE 2.

NORMAL THRESHOLDS (AVERAGE OF EIGHT EARS)

(Db Above 0.0002 Microbar)

Overall Noise Level		250	500	1000	2000	3000	4000	6000	8000
Quiet.....	M	37	18	12	13	18	20	28	27
	SD	4	4	2	4	4	6	9	6
70 Tape.....	M	51	49	55	52	54	49	50	47
	SD	5	2	2	3	3	3	4	4
70 Generator...	M	52	49	54	53	56	52	54	44
	SD	3	2	3	2	0	2	3	3
100 Tape.....	M	81	82	86	84	89	84	86	82
	SD	3	3	3	3	4	2	5	5
100 Generator...	M	78	81	85	83	86	85	91	81
	SD	2	3	2	2	3	3	3	3
110 Tape.....	M	95	92	96	94	97	92	95	91
	SD	2	3	0	3	4	2	4	5
110 Generator...	M	89	92	96	95	98	97	96	91
	SD	4	3	0	3	3	2	3	3

Hawkins and Stevens curves in this figure are about 2 db lower than those corresponding in the present experiment.

RESULTS.

The individual results of our measurements on 81 pathological ears are given in Table 3. All of the thresholds in this table are expressed as db above 0.0002 microbar. The reader may convert these to hearing loss by subtracting from them the sound pressure levels corresponding to 0-db hearing loss given in Table 1 (masked thresholds should be corrected by levels "with network"). The first eight columns in Table 3 give the thresholds for eight frequencies measured in the quiet. The last eight columns give the thresholds for the same frequencies in the presence of the 100-db noise.

Some of the thresholds in this table are followed by an asterisk (*). This symbol signifies a quiet threshold that is more than 5 db greater than the threshold at that frequency for a normal listener in the presence of the noise. We have omitted these thresholds from our summary calculations, presented in Table 4, because they represent quiet thresholds that could not be masked by this noise. In other words, we have omitted from our calculations all quiet thresholds that are so high that the noise did not change the threshold substantially.

In the last column of Table 3 a set of code numbers is used to indicate the criteria that were used to assign the patient to one of our six diagnostic categories. The explanation of these code numbers is as follows:

1. Bone conduction (B.C.) normal, 0-20 db.

2. B.C. better than Air Conduction (A.C.) but not normal.

3. B.C. same as A.C.

4. No recruitment by loudness balance.

5. Recruitment by loudness balance.

6. Discrimination score for PB words 80 per cent or higher.

7. Discrimination score for PB words between 60 to 79 per cent.

8. Discrimination score for PB words less than 60 per cent.

9. Conductive impairment confirmed by surgery.

Those patients for whom neither *4* nor *5* is indicated were not examined for recruitment. Where we used loudness balance, it was with an alternate binaural judgment at the same frequency or a monaural judgment between two different frequencies. Too much space would be required to indicate all of this diagnostic information quantitively, so we have used this code to indicate grossly what measurements, in addition to the masking tests, were used and what the diagnostic indications were.

TABLE 3.

INDIVIDUAL THRESHOLDS.

(Db Above 0.0002 Microbar)

Ear	In Quiet								In 100 DB Noise								Diagnosis (See Text for Key)
	250	500	1KC	2KC	3KC	4KC	6KC	8KC	250	500	1KC	2KC	3KC	4KC	6KC	8KC	
ED/R	65	48	44	47	56	63	72	73	84	83	91	87	91	83	88	84	1,6,9
AMB/L	95*	83	79	57	71	78	92*	..	89*	88	91	77	86	83	..	..	1,7,9
ET/L	85*	83	79	77	66	68	72	83	89*	88	86	87	86	83	83	84	1,6
WR/L	85	78	69	77	71	78	77	63	84	83	86	82	86	83	83	79	1,7
RR/R	80	68	59	57	61	58	77	73	79	83	91	82	81	78	83	79	1,7
RR/L	90*	83	64	67	71	73	67	68	89	83	81	82	81	73	78	79	1,7
FW/L	65	53	49	37	46	48	52	53	79	78	86	87	86	83	83	84	1,6
BB/R	70	58	49	42	56	53	77	73	79	83	86	82	81	78	78	74	1,6,9
BB/L	70	63	54	52	86	68	77	78	84	83	86	82	86	83	83	84	1,6,9 Conductive
PM/R	85*	68	59	42	46	38	52	48	89*	83	86	82	86	78	83	74	1,6
PM/L	85*	53	49	42	36	43	52	48	89*	78	86	82	86	83	83	74	1,6
HB/L	80	58	49	42	41	48	42	48	79	78	81	82	86	78	83	74	1,6
AH/R	70	63	49	42	51	48	77	88*	79	83	81	82	81	83	88	89*	1,6,9
AH/L	85*	83	64	57	61	58	77	98*	84*	88	86	82	81	83	88	94*	1,6
RM/L	80	58	59	62	51	43	57	68	84	78	86	82	81	83	78	79	1,6
LW/R	55	38	34	47	26	28	37	28	79	78	81	82	81	83	83	79	1,4,6
MF/L	50	43	49	27	36	48	57	58	79	83	86	87	86	83	83	79	1,3,6
MF/R	70	63	59	47	51	43	57	63	79	83	86	82	91	83	83	89	1,3,6,6,9
RM/R	75	73	54	52	81	93*	97*	88*	84	83	86	82	101	93*	98*	84*	2,4,6,9
JW/R	100*	83	89	72	76	78	92*	83	109*	88	96	87	86	88	88*	89	1,4,8
VG/L	90*	68	59	57	46	73	77	88*	89	83	86	82	81	78	83	89*	2,6
WE/R	90*	78	74	72	66	68	77	63	94*	88	91	92	86	83	88	79	2,7
WE/L	90*	73	64	67	76	83	77	58	94*	83	86	82	86	83	83	74	1,7
JB/R	65	43	44	42	51	68	87	103*	89	83	91	92	86	83	93	99*	1,4,6,9
JB/L	85	68	49	47	46	73	72	78	89	83	86	87	86	83	83	84	1,6
WR/R	80	73	69	57	61	63	62	53	84	83	86	82	86	83	83	74	2,6
IG/R	110*	98*	94*	82	81	78	67	78	104*	98*	96*	87	86	83	78	84	2,6
BG/R	70	58	49	47	36	43	42	48	79	78	81	82	81	78	83	79	1 3,6
BG/L	50	38	29	27	36	38	42	43	79	73	81	82	86	83	78	74	1,3,6,9
RW/R	90*	78	79	67	66	68	67	88	94*	88	91	87	91	88	88	94	2,6
RW/L	80	63	69	77	76	78	82	..	84	83	86	87	91	83	83	..	1,7,9
EK/R	80	58	54	47	41	48	37	53	84	83	91	87	86	83	78	79	1,3,6 Conductive-Mixed
EK/L	85	68	54	52	51	53	67	68	89	83	86	87	86	78	83	79	1,6
ED/L	90*	68	59	62	66	78	92*	93*	94	83	86	87	86	83	93	94	2,6
DS/R	70	58	59	57	51	48	57	63	84	83	91	87	86	78	83	84	1,6
DS/L	90*	73	69	62	66	58	57	53	94	83	86	82	86	78	78	79	2,6
CO/R	105*	88*	89	72	71	78	72	73	109	88	91	87	86	83	83	84	2,7,9
CO/L	95*	73	69	67	71	63	57	68	99	83	86	82	86	78	78	79	2,6
AMB/R	90*	88*	84	72	71	93*	82	88*	94*	88*	86	77	86	93*	83	89*	2,6
FL/R	85*	68	69	72	76	68	82	88*	89*	83	86	87	91	88	88	94*	2,6
FL/L	80	73	64	67	76	73	102*	98*	94	83	86	87	91	88	..	104*	2,6
ET/R	85*	83	69	67	66	73	87	98*	84*	88	81	82	86	83	88	99*	2,6
LL/R	85*	78	74	72	71	68	72	73	89*	83	86	87	86	88	93	79	2,8
LL/L	90*	78	74	67	66	63	67	58	94*	83	86	87	86	88	88	74	1,8
MD/R	80	68	64	67	71	63	77	83	84	78	86	87	86	83	88	89	2,4,6,9
MD/L	50	38	34	52	36	48	57	63	74	78	86	87	86	88	88	79	1,6

FL/R	85*	68	69	72	76	68	82	88*	89*	83	86	87	91	88	88	94*	2,6	
FL/L	80	73	64	67	76	73	102*	98*	94	83	86	87	91	88	..	104*	2,6	
ET/R	85*	85	69	67	66	73	87	98*	84*	88	81	82	86	83	88	99*	2,6	
LL/R	85*	78	74	72	71	68	72	73	89*	83	86	87	86	88	93	79	2,8	
LL/L	90*	78	74	67	66	63	67	58	94*	83	86	87	86	88	88	74	1,8	
MD/R	80	68	64	67	71	63	77	83	84	78	86	87	86	83	88	89	2,4,6,9	
MD/L	50	38	34	52	36	48	57	63	74	78	86	87	86	88	88	79	1,6	
WK/R	80	73	69	67	76	108*	102*	93*	84	83	86	82	86	108*	..	94*	2,7	
WK/L	80	78	74	97*	91	98*	102*	..	84	83	91	102*	96	98*	..	..	2,7	
RG/R	95*	83	74	72	76	73	97*	103*	99*	88	86	82	86	88	83*	89*	2,7	
VG/R	75	68	59	57	51	68	87	93	84	78	86	87	86	83	88	89*	2,6,9	Mixed Nerve
BM/R	75	53	54	57	66	78	97*	93*	84	78	81	82	86	98	98*	94*	3,7,9	
BM/L	70	58	54	57	86	98*	..	..	84	83	81	82	96	98*	..	..	2,6,9	
JS/L	40	23	14	22	21	23	47	33	79	83	86	87	91	88	93	84	3,5,6	
CN/L	95*	88*	84	72	76	68	72	73	99*	98	91	87	91	83	88	89	3,5,8	
JL/R	35	23	15	26	24	31	57	60	89	83	91	87	91	88	88	84	3,5,6	
WL/L	60	43	19	27	56	63	57	68	84	83	91	87	91	88	83	89	3,5,6	
MM/L	40	23	14	17	31	73	87	103*	89	88	91	87	91	88	88	99*	3,5,6	
FH/R	80	73	64	62	56	58	67	68	94	93	86	87	86	83	83	79	3,5,8	
FB/R	80	68	44	32	36	43	62	73	84	83	86	82	81	93	83	79	3,5,8	Nerve with
BW/L	30	13	4	27	56	73	72	58	79	83	81	87	91	88	88	74	3 5 6	Recruitment
CM/R	40	18	9	12	81	88	82	73	79	78	86	87	91	93	93	89	1,3,5,7	
CM/L	35	13	9	12	66	63	37	23	79	78	86	87	86	88	88	74	3,5,6	
AT/R	60	48	34	57	81	78	72	73	84	78	81	82	86	83	93	89	3,5,8	
AO/L	55	33	24	32	21	28	47	58	89	83	86	82	86	88	83	79	3,5,6	
EB/R	50	38	24	37	46	73	67	63	79	83	81	82	86	83	88	79	3,5,7	
LR/R	50	38	24	22	66	78	87	83	79	78	81	82	86	88	93	89	3,5,6	
AB/L	85*	83	74	52	71	68	82	83	94*	88	91	87	91	93	88	79	2,6	
WB/R	80	63	54	57	76	83	..	..	84	78	86	82	86	83	..	..	3,4,6	
WB/L	50	23	19	27	31	58	67	93	79	78	86	82	86	83	83	94*	3,6	Nerve No
LR/L	80	58	54	72	76	88	97*	108*	99	88	86	82	91	98	93*	..	3,4,8	Recruitment
RT/L	95*	78	..	..	..	..	..	..	99*	88	..	..	..	..	..	..	3,4,8	
DH/L	40	33	29	27	41	43	37	33	94	88	91	92	91	88	73	74	3,6	
AG/R	30	13	19	77	81	73	77	63	74	73	86	87	91	88	98	89	3,8	
AG/L	30	18	44	77	81	73	67	73	74	73	86	87	86	83	88	84	3,8	
RG/L	35	13	14	17	31	48	42	33	84	83	86	87	91	88	83	79	3,6	
WL/R	30	18	9	22	56	53	62	48	84	88	91	92	91	88	88	84	3,6	
SH/R	40	33	24	22	26	43	47	58	79	78	86	87	91	88	83	79	3,6	Nerve, Recruit-
FH/L	50	43	34	27	46	48	67	68	79	83	81	87	86	88	83	74	3,6	ment not explored
MM/R	45	23	14	17	21	33	47	68	84	83	91	87	86	88	93	94	3,6	
JL/L	40	23	15	16	19	26	62	45	84	83	86	87	91	88	88	84	3,6	
BW/R	30	18	4	17	16	58	67	53	79	78	86	87	86	88	93	79	3,6	
MU/L	70	58	39	12	31	48	72	93*	84	83	86	82	91	88	93	89*	3,7	

The following

JL/L	40	23	15	16	19	26	62	45	84	83	86	87	91	88	88	84	3,6	
BW/R	30	18	4	17	16	58	67	53	79	78	86	87	86	88	93	79	3,6	
MU/L	70	58	39	12	31	48	72	93*	84	83	86	82	91	88	93	89*	3,7	
The following measurements made at 110 db noise:																		
PB/R	75	83	84	87	96	83	..	..	94	93	96	97	96	88	..	..	2,7	
PB/L	85	88	84	87	..	113*	102*	108*	89	88	96	92	..	103*	..	..	2,8	
PE/R	80	73	74	102*	96	98*	97	98*	89	88	96	102*	101	103*	93	99*	2,7	Mixed Nerve
PE/L	65	63	59	82	91	88	92	98*	94	93	96	102	101	98	98	99*	2,7,9	
SH/L	..	..	..	92	96	88	82	83	..	..	..	92	101	93	93	94	3,5,8	Nerve with
MU/R	115*	103*	99	87	86	88	92	108*	109*	93*	101	97	101	103	98	104*	3,5,8	Recruitment
AB/R	100*	103*	104*	102*	101	93	..	103*	104*	98*	106*	102*	101	98	..	..	2,4,8	Nerve, no Recruitment

In general, what we have called the *conductive* cases are characterized by normal bone-conduction thresholds and very good discrimination for speech. Mostly these were cases of otosclerosis many of which had been fenestrated before these tests. Others sustained conductive impairment either as a result of middle-ear infection or atresia. The group that we have called *conductive-mixed* are essentially the same except that we detect by various means somewhat more nerve involvement than in the first group. When this nerve component became so large as to appear more responsible for the general deafness than the conductive component, we labeled those cases *mixed-nerve.*

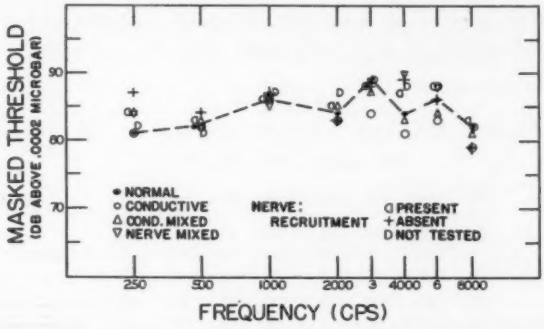


Fig. 3. Pure-tone thresholds at eight frequencies measured in the presence of white noise (20 to 7000 cps) at an overall level of 100 db above 0.0002 microbar. Each symbol represents the average threshold for each diagnostic group detailed in Table 3 plus the normal. These data may indicate some systematic difference at 3000, 4000, and 6000 cps. Here the thresholds for the conductive and conductive mixed groups are constantly below those of the other groups. Only the extreme differences are significant, however, and the total range lies within 10 db.

Whenever the hearing loss for air conduction was essentially the same as that for bone conduction, the case was placed into one of three categories of *nerve deafness*:

1. Cases that showed *no recruitment* by the loudness-balance test. One (AB) sustained a multiple skull fracture that resulted in bilateral damage to VIth, VIIth, and VIIIth cranial nerves, and another gave a history of hereditary deafness.

2. Cases that showed *recruitment* on the loudness-balance test. There is evidence of cochlear degeneration, Meniere's disease, or acoustic trauma.

3. Cases in which *recruitment* was *not examined.* Many of these are the opposite ears from those categorized under *1* or *2.*

Table 4 summarizes the results for pure-tone thresholds measured in the presence of 100 db overall noise for each of the six diagnostic categories and for normals. These results are also presented graphically in Fig. 3. It is clear that the masked thresholds at all eight frequencies are very nearly the same for all diagnostic categories and for the normal. We conclude, therefore, that differences among the masked thresholds in such a noise for different patients are not reliable indicators of any particular type of pathology.

TABLE 4.

AVERAGE MASKED THRESHOLDS (IN 100 DB NOISE).

(Db Above 0.0002 Microbar)

Frequency	250	500	1000	2000	3000	4000	6000	8000	Diagnosis
Mean	81	82	86	84	89	84	86	82	
SD	3	3	3	3	4	2	5	5	Normal
No. Cases..	8	8	8	8	8	8	8	8	
Mean	81	82	86	83	84	81	83	79	
SD	3	4	3	2	3	8	3	4	Conductive
No. Cases...	10	16	16	16	16	16	15	13	
Mean	84	83	87	85	87	83	84	81	
SD	5	3	3	3	4	3	4	4	Conductive Mixed
No. Cases...	15	27	29	30	30	28	26	21	
Mean	84	82	85	83	89	90	88	..	
SD	0	4	4	2	5	8	..	..	Mixed Nerve
No. Cases...	5	6	6	5	6	3	1	..	
Mean	87	84	87	83	88	89	86	79	
SD	10	5	2	2	3	7	..	..	Nerve, No Recruitment
No. Cases...	3	5	4	4	4	4	2	1	
Mean	84	83	86	85	88	87	88	83	
SD	5	4	4	2	3	3	4	6	Nerve, With Recruitment
No. Cases...	13	13	14	14	14	14	14	13	
Mean	82	81	87	87	89	88	88	82	Nerve Recruitment not explored
SD	6	5	3	3	2	1	7	6	
No. Cases...	11	11	11	11	11	11	11	10	

SUMMARY.

A review is given of papers on noise audiometry that report differences among the masked thresholds of several groups of patients, each group representing a different diagnosis. A brief review is given of the methods by which the intensity of a noise can be measured and specified. It is pointed out that theoretically, particularly with respect to the concept of the critical band, the masked thresholds for patients with different kinds of hearing loss should be the same providing that the quiet threshold is not higher than the normal masked threshold. Experimental data are reported which show that the masked thresholds for pure tones ranging in frequency from 250 to 8000 cps taken in the presence of a white noise whose overall level between 0 and 7000 cps is 100 db (re 0.0002 microbar) all lie very close to one another, mostly within a 5 db range and at higher frequencies within a 10 db range. It is concluded that the masked threshold is not a useful indicator for differential diagnosis.

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BOOK REVIEW.

DYSARTHRIC SPEECH (Speech in Cerebral Palsy). By Emil Froeschels, M.D., President, International Society for Logopedics and Phoniatrics; President, New York Society for Speech and Voice Therapy, Inc., Physician in charge of the Speech-Voice Clinic, Beth David Hospital, New York City, N. Y. 172 pages with Index and 34 figures. Magnolia, Mass: Expression Company, 1953. Prices $3.75.

This book is intended to furnish information to "scientists and laymen" in the neuromuscular relationships in dysarthritic speech and the therapy therefor. It contains the author's previously stated therapeutic approach based on the "chewing" method in which the patient is directed to make chewing movement which eventually are combined with phonation.

S. R. S.

THE EFFECT OF EFOCAINE IN THE CONTROL OF POSTOPERATIVE PAIN.*

ROLAND F. MARKS, M. D.,
Walnut Creek, Calif.

The purpose of this report is to present observations made following use of Efocaine.† It is my opinion that its effect upon tissue must be considered carefully.[1]

I report fourteen cases in which Efocaine was used in an effort to relieve postoperative tonsillectomy pain as suggested by Risdon.[2] The method was to inject 1 c.c. at the base of the tongue and attachment of the posterior pillar, the base of the tongue, and the attachment of the anterior pillar. The cases were nine general and five local. The primary infiltration preoperatively for all cases, including local and general, was approximately 6 c.c. of 2 per cent Zylocaine with epenephrine 1-50,000, using the usual four cardinal points of injection bilaterally. The injection of the Efocaine was made after hemorrhage had been controlled and the dissection of tonsil completed. In one local case injection of approximately 4 cc. bilaterally was made and an immediate profuse, severe edema of the soft palate occurred, resulting in complete obstruction of the postnasal space. Fortunately, this did not extend into the hypo-pharynx, but emergency tracheotomy was anticipated if necessary. The edema resolved in approximately four hours; intravenous benedryl, 30 milligrams, was administered, and 50 milligrams orally q3h for twenty-four hours ordered.

The postoperative results were as follows: *1.* Fourteen cases stated marked relief from postoperative pain; one of these stated that discomfort was less than pain from acute

*Read before the Alameda-Contra Costa County Medical Society, May 19, 1953.

†Formula Efocaine: Procaine, 1 per cent; Procaine Hydrochloride, 0.25 per cent; Butyl-p-aminobenzoate, 5 per cent; Polyethylene-glycol-300, 2 per cent; Propylene Glycol, 78 per cent; and water for injection, 20 per cent. Preserved with Sodium Metabisulfite 0.1 per cent and Phenyl Mercuric Borate (1:25,000).

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tonsillitis which had occurred six weeks previously; postoperative pain controlled for approximately four to five days; however, more severe in three cases than usual. *2.* Six cases had moderate to severe hemorrhage from sixth to twenty-fourth day. One case of recurring hemorrhage on twelfth day required multiple suturing at base of tongue to control profuse arterial hemorrhage. *3.* Large pale slough, approximately one centimeter in diameter, noted at base of tongue at site of injection on seventh day postoperative in almost all cases. *4.* One case had increased pain following injection which was also accompanied by severe hemorrhage. In five cases the hemorrhage was bilateral..

The relief of pain following tonsillectomy was in most cases excellent; however, the postoperative hemorrhage and necrosis does not justify its use following tonsillectomy. Since observing the above, Moore[3] has reported the necroses following injection into monkeys. According to his report, there is perineuritis or complete destruction of the nerve injected.

SUMMARY.

1. As a result of clinical observation and animal experimental study, the use of Efocaine must be carefully evaluated and its dangers realized fully. *2.* Edema delayed healing and postoperative hemorrhage are the immediate complications.

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2. RISDON, T. ALLEN, M. D.: *A. M. A. Archives of Otolaryngology,* 57:86.

3. MOORE, DANIEL, M.D.: Personal communications and report to Conference in Anesthesiology, Los Angeles, Calif., 1953. Complete publication to appear at later date.

1806 Bonanza Street.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

September 1, 1953.

Audicon Models 400, 415, 530 and 615.
Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 149 Church St., New York 7, N. Y.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Normatone Model C.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom 5.

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp.

Silvertone Model J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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New fire-proof buildings beautifully located opposite Forest Park. Modern Dormitories and Equipment. Best home environments. Pupils constantly in care of teachers or experienced supervisors.

ORAL SCHOOL FOR DEAF CHILDREN

C. I. D. offers all advantages of exclusively Speech Training and expert medical supervision for both Resident and Day Pupils.
Nursery School (2 years of age) through the Elementary Grades.

ACOUSTIC TRAINING FOR CHILDREN WITH RESIDUAL HEARING

Salvaging of Residual Hearing is a specialty of C. I. D. The Acoustic Method was created here. Group and individual hearing aids used for class instruction at all grade levels.

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Private and Class Instruction for Hard-of-Hearing Adults and Children.
Conversational Classes for advanced pupils. Speech conservation stressed.

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Private and Class Instruction for children with normal hearing and delayed speech or defective speech.
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